RESEARCHES UPON THE PATHOLOGY

OF

SUBDURAL MEMBRANE FORMATION.

GRADUATION THESIS PRESENTED TO THE UNIVERSITY
OF EDINBURGH FOR THE DEGREE OF DOCTOR OF MEDICINE
BY

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30TH APRIL, 1895.

Note. A box containing 205 microscopic specimens is produced with this thesis.

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PRELIMINARY NOTE

As an account of a portion of the researches which form the subject of this thesis has already been published in a paper written conjointly with another author(156), it is necessary that I should here explain my position with regard to that publication. The article referred to, which appeared in the Edinburgh Medical Journal for February last, is one of a series which is being published by Dr James Middlemass, senior assistant physician at the Royal Edinburgh Asylum, and myself on "The Pathology of the Nervous System in relation to Mental Diseases." When drawing out our scheme for this undertaking we assigned to each certain sections of it, the whole of the work required for which, up to the writing of the draft of the paper, was to be undertaken by the one to whom it was allotted. The section on morbid conditions of the dura mater was among those which were assigned to me. I therefore had to do the laboratory work required for the article referred to, to study the previous literature of the subject, and to write the draft of the paper. A large number of the more important microscopic specimens were submitted to Dr Middlemass for examination, and we freely discussed the interpretation of the various appearances which they presented.

From time to time we also debated at considerable length the various difficult problems which the subject presented for solution. How much I owe to this aid it is impossible for me to estimate, but it is certain that something must be deducted on account of it from any credit which may seem to accrue to me from the researches recorded in this thesis. Since the publication of our conjoint paper in February last I have resumed independently the study of the pathology of subdural membrane formation. I have re-examined all the cases which I had previously studied, preparing many new microscopic specimens from each of those which are included in the series here described. Some of my earlier cases have been excluded on account of sufficient material not having been preserved for the preparation of specimens of the kind required for the investigation, and I have investigated many entirely new cases. As the result of these more recent researches I have a large number of entirely new observations to add to those recorded in the published paper, as well as many new conclusions to deduce from them.

When I first took up the study of this subject I used only transverse sections of the dura and stripped preparations of thin false membranes, and I failed to make anything of it. I was convinced,

however, that the all-important changes took place at the inner surface, and it occurred to me some months ago that if this could be examined in horizontal view the problem of the pathology of subdural membrane formation might be solved. I therefore devised a method of cutting what I call "superficial horizontal sections." By means of these extensive areas of the inner surface of the dura may be examined with high powers, and morbid changes may be observed which are not recognisable in transverse sections. I have examined by this method a series of duras from the insane and from general hospital patients, and the observations which I have made upon them, and the conclusions to which they have led me constitute the bulk of the original matter contained in the succeeding pages. I have made no attempt to classify the cases of which descriptions are given, and they are arranged merely in the order in which they were studied for this thesis, except that the general hospital cases have been placed at the end of the series. All the cases from the insane came under my own observation in the course of my work as pathologist at the Royal Edinburgh Asylum, with the exception of No. 24 which is introduced on account of its special interest.

The method by which I prepare superficial horizontal sections of the dura is as follows.

After hardening in potassium bichromate solution, square pieces about half an inch in diameter are cut with scissors and allowed to soak in water for an hour. A few drops of a thin solution of dextrine in water, to which some carbolic acid has been added, are then placed upon the section-plate of an ether freezing microtome and firmly frozen. A perfectly level surface is next cut with one sweep of the section knife. A piece of the dura is then taken up with forceps and, after superfluous water has been removed from it by laying the outer surface on a piece of filter paper or a towel, is laid with the inner surface undermost upon the smooth frozen surface. The ether spray is at once again put in action so that the piece of tissue also is frozen. The section-plate is next slightly lowered and successive slices of the dura are cut away until only a thin layer of tissue remains on the surface of the dextrine. The section-plate is then raised so as to place this surface considerably above the sweep of the knife with which a thick slice is next cut and placed in a bowl of water, of course carrying with it the required section of the dura, which is ready for staining a few minutes afterwards. The chief points of importance are to have a perfectly level firmly frozen dextrine surface, and a sharp knife.

After some practice it is quite possible to cut sections of this kind sufficiently thin for satisfactory examination with high powers. One has often to be content, however, with preparations of which only a portion is suitable for this purpose. The staining which I find most satisfactory is that with haematoxylin and eosine. At first I used almost exclusively alum carmine and eosine, for the reason that even somewhat thick sections stained in this way are sufficiently transparent to be examined with high powers. With practice, however, I have been able to cut much thinner sections than I could formerly obtain, and I have found that with these haematoxylin and eosine gives very much superior preparations. Indeed the endothelial changes which occur cannot be distinctly recognised with alum carmine and eosine staining. This is the explanation which I have to give of the fact that these endothelial changes,- which as I shall presently endeavour to show are of the utmost importance,- passed practically unrecognised in the course of my earlier researches and are not described in the paper in Edinburgh Medical Journal. In mounting these superficial horizontal sections care must be taken that the inner surface is turned towards the coverglass. The stained section should be placed upon a slide and examined without a coverglass with the low power

of the microscope by means of which it is usually easy to tell which surface is uppermost. For some purposes, however, it is of advantage to have the outer or cut surface turned upwards.

The number of cases upon which my conclusions are based is certainly smaller than might have been desired. Not only, however, is the preparation of these superficial horizontal sections laborious work, but it must be remembered also that in studying them I have been entering upon a new pathological field the phenomena to be observed in which are of the most varied kinds and many of them in a high degree difficult to interpret. Consequently the preparation and examination of specimens from a single case have in many instances entailed several days of labour. Some representative specimens have been selected from each case and are submitted for examination with this thesis. Special spots to which attention is directed are marked on the under surface of the slide with an ink-line which may be followed with the low power of the microscope. I believe that these preparations will carry with them convincing evidence of the important results which may be obtained by the employment of superficial horizontal sections. I have only used them in this research,- and in a conjoint one with Dr James Middlemass upon the morbid conditions of the pia-arachnoid (see

Edinburgh Medical Journal for April and May),- but I would suggest here their employment also for the study of morbid changes in other smooth surfaces, such as those of the peritoneum, pleura, pericardium, joint cavities, arteries, veins, the mouth and oesophagus.

I have to express my great indebtedness to Dr G. M. Robertson for very kindly giving me access to a large number of French and German monographs, as well as other literature on the subject of this thesis, which he has collected. I have also to acknowledge another debt of a different, but even more important kind which I owe him, in as much as it was his most thoroughly scientific and suggestive papers on the subject which first aroused my interest in the pathology of subdural membrane formation. I have to thank Dr R. F. C. Leith and Dr Robert Muir for the material obtained from the pathological department of the Royal Edinburgh Infirmary.

LITERATURE UPON SUBDURAL MEMBRANES.

1760.

1. Morgagni. "De sedibus et causis morborum."

1812.

2. Nepple. "Diss. de physiologie pathologique sur les fausses membranes et les adherences". Thèse de Paris.

1817.

3. Houssard. Bibliothèque Medicale, LV, p.69.

1818.

4. J.Abercrombie. "Observations on chronic inflammation of the brain and its membranes."

1819.

5. Serres. Annuaire des hôpitaux civils de Paris.

1823.

6. Rostau. "Recherches sur le ramollissement du cerveau."

1826.

7. Calmeil. "De la paralysie générale chez les aliénés."

8. Bayle. "Traité des maladies du cerveau et de ses membranes," p.250.

1827.

9. Wardrop. "Singular appearance of the dura mater of a maniac." Lancet, XII, p.255.

1834.

10. Andral. Clinique med., Paris.

11. Baillarger. Arch. gén. de med.

1835.

12. Velpean. "Dure mère,- Pathologie,"- Dict. de méd., Paris. X., p.514.

13. Longet. Thèse de Paris.

1836.

14. Lelut. "Memoire sur les fausses membranes." Gaz. méd. de Paris, IV., 29.

15. Baillarger. Thèse de Paris.

1837.

16. Baillarger. "Du siège de quelques hémorrhagies menigées." Arch. gén. de méd.

1838.

17. Boudet. "Memoire sur l'hémorrhagie des meninges." Journ. des connaiss. méd.

1842.

18. Legendie. "Memoire sur les hémorrhagies dans la cavité de l'arachnoid pendant enfance." Rev. méd. franc. et étrang., Paris IV., 344.

19. Rilliet et Barthez. Gaz. Med.

1843.

20. Aubanal. "Des fausses membranes de l'arachnoide et principalement de leur mode de formation chez les aliénés." Ann. Méd.-psych., Paris. II., 55,201.

1844.

21. Rokitansky. Spec. Path. Anat.,- Sydenham Society's Trans., I.

1845.

22. Prescott Hewitt. Medico - Chirurgical Transactions. Also Medical Times, I., p.452.

23. Prus. "Memoire sur les deux maladies connues sous le nom d'apoplexie menigée." Mem. de l'Acad. Roy. de Méd.

1849.

24. Schubzenberger. "De l'hémorrhagie dans la grande cavité arachnoidienne." Gaz. méd. de Strassb., IX., 129.

1850.

25. Coote. "On the formation of adventitious membranes in the sac of the arachnoid." Med. Times, I., 306.

1852.

26. Bauchet. "Kyste hématique de la dure-mère."

Bull. Soc. Anat. de Paris, XXVI., 113.

1853.

27. Bricheteau. "Kyste hémorrhagique de la cavité arach." Gaz. de hôp., Paris, XXVI., 368.

1855.

28. Hesehl. Comp. der allg. und spec. path., Wien.

29. Hasse. Handb. d. spec. path. von Virchow, IV.

30. Flower. "Encysted coagulation in the arachnoid cavity." Trans. Path. Soc. Lond., VII.

31. Quain. "Arachnoid Cysts." Trans. Path. Soc. Lond., VI.

32. Durand - Fardel. "Krankheiten d. greisalters."

1856.

33. Virchow. "Hœmatoma duræmatris," Verhandl. d. med.- physik. Gesellschaft zu Wurzburg., Bd.VII.

34. Cruveilhier. Atlas d'Anat. path.

1857.

35. J. W. Ogle. "The formation of false membranes," Arch. med. Lond. I., 270.

1859.

36. Schuberg. Arch. f. path. anat., XVI., 464.

37. Brunet. "Recherches sur les néomembranes et les kystes de l'arachnoide." Paris.

38. Hasse. Handb. d. spec. path. d. Virchow.

1860.

39. J. W. Ogle. Arch. Med. Lond., II., 85.

40. Weber. Arch. d. Heilk. I.

41. Chareot et Vulpean. Gaz. hebdom., p.729

1861.

42. Schuberg. Arch. f. path. anat., XXI., 301.

1862.

43. Lancereaux. Arch. gén. de Med.

44. Griesinger. Arch. de Heilk. p.33.

45. Marcé. "Traité des maladies mentales." p.465.

46. Ramaer Virchow's Arch. Bd. XIV.

1863.

47. Brunet. Gaz. des hôp.

48. Pirotais. De la pachy. hémor., Thèse de Strasbourg.

49. Perroud. "Hémorr. dites intra-arach.," Gaz. Méd. de Lyon, XV., 492.

50. Pauvert. Thèse de Paris.

1864.

51. J. W. Ogle. "Blood-cysts situated within the arachnoid cavity in cases of general paralysis of the insane." Jour. of Ment. Sc., p.525.

52. Christian. "Étude sur la pachyméningite hémorrhagique." Strasbourg.

53. Laborde. "Contrib. a l'étude des conditions

path. des kystes sang. de l'arach." Compt. Rend. Soc. d. Biol., Paris, I., 70.

1865.

54. S. Wilks. "Cysts in the arachnoid cavity." Jour. Ment. Sc., p.94.

55. Lioville. "Hématomes de la dure-mère." Bull. Soc. Anat. d. Paris, XL., 673.

56. Breyton. "Obs. de pachyméningite avec hématome." Mem. et Compt. Rend. Soc. d. Sc. Méd. d. Lyon., IV., 60.

57. Judée. "Obs. de pachyméningite." Mem. et Compt. Rend. Soc. d. Sc. Méd. d. Lyon., IV. 271.

58. Do. Gaz. Méd. d. Lyon, XVII., 149.

59. S. Wilks. "Arachnoid Cysts." Trans. Path. Soc. Lond.

60. Regnard. Gaz. d. Hôp., Paris, 37.

1866.

61. Gorry. "Etude sur l'étiologie de la pachymen. hémorrh." Paris.

1867.

62. Blachez. Union Médicale.

1868.

63. Kremiansky. Arch. f. path. anat. XLII., p.129.

64. S. Wilks. Med. Times and Gazette.

65. J. Pons. "Sur la Pachyméningite." Paris.

66. Botentuit. "Pachyméningite." Bull. Soc. Anat. de Paris, XLIII., 303.

67. Joffroy. "Pachyméningite." Bull. Soc. Anat. de Paris, XLIII., 435.

68. Kessler. "Beitrag zur diagnose des hœmatom der dura mater." Berlin.

1870.

69. Dickson. "Dura mater of the insane." Trans. Path. Soc. Lond., XXI.

1871.

70. H. Sutherland. "Arachnoid Cysts." West Riding Asylum Reports. I., 218.

71. Sperling Central. f. med. wiss., Berlin. IX., 449.

1872.

72. Sperling. "Ueber pachymeningitis haemorrhagica." Konigsberg.

73. Hammond. "Haematoma of the dura mater." Quart. Jour. Psycholog. Med., New York, VI., 646.

74. Landouzy. "Macrocephalie, pachyméningite et fausse memb." Bull. Soc. Anat. de Paris, XLVII., 520.

75. Hanot. "Sache de l'arachnoide observe dans un

cas de paralysie générale." Bull. Soc. Anat. de Paris. XVII., 154.

1873.

76. Rindfleisch. Path. Hist., II. London.

77. McCormick. "Haematoma of the dura mater." American Practitioner, Louisville, VIII., 224

78. Behier. "Cas de pachyméningite." Gaz. hebdom. de med. X., 148.

79. Laborde. Compt. Rend. d. l. Soc. d. Biol., p. 257.

80. Luneau. "Path. des hem. prim. de la cavité de l'arach. cranienne." Thèse de Paris.

1874.

81. Christian. "Nouvelles observations de pachyméningite chez les aliénés." Ann. Med. Psycholog.

82. Bruberger. Virchow's Archiv. p. 215.

1875.

83. Crichton Browne. "Arachnoid Cysts." Journ. of Psycholog. Med.

84. Paulus. "Verkalkung und Verknockerung d. Hämatomes d. Dura Mater." Erlangen.

85. Landouzy. "Pachymén. sur un nouveau né." Bull. Soc. Anat. de Paris, L., 388.

1876.

86. Lawson. Brit. and For. Med. Chir. Review.

87. Thorogood. "Case of haematoma of the dura mater." Lancet I., 738.

88. Hondoux. "Contrib. a l'étude de la pachyméningite hémorr." Nancy.

89. Bauduy. "Pachymeningitis." St. Louis Clin. Rec. II., 265.

90. Jequin. Med. Rec. of New York. XI., 163.

91. Laborde. Compt. Rend. de la Soc. de Biologie, p. 181.

1877.

92. Huguenin. Ziemssen's Cyclopaedia of Medicine. XII., p. 386.

93. Greenfield. Lancet, II., p. 767.

94. Althaus. "Diseases of the nervous system." p. 186.

95. "Discussion sur les pachyméningites," Bull. de la Soc. de Méd. Ment. de Belg., XII., 21.

96. Carafi. "Pachyméningite avec hématome de la dure-mère." Bull. Soc. Anat. de Paris, LII., 325.

97. Furstner. "Zur genese und symptomatologie der pachymeningitis haemorrhagica." Arch. f. Psychiat., Berlin, VIII., 1.

98. Shaw. "Traumatic cerebral pachymeningitis." N.Y. Med. Jour. XXVI., 612.

99. Wilson. Trans. Path. Soc. Philadelphia, VI., 126.

100. Clouston. "On the pathological significance of false membranes under the dura mater in insanity." Journ. Ment. Science. p. 349.

1878.

101. Eulenberg. Lehrbuch der Nerven-Krankheiten.

102. " "Discussion sur les pachyméningites," Bull. de la Soc. de Méd. Ment. de Belg., XIV., 15.

103. Lancereaux. "Notes sur quelque faits de pachyméningite gommeuse." Bull. Acad. de Med., Paris, VII., 901.

104. Landouzy et Remy. "Pachyméningite avec hématome comprimant les regions corticales non motrices." Bull. Soc. Anat. de Paris, LII., 510.

105. Rosenthal. Traité des maladies du systeme nerveux.

1879.

106. Voisin. Traite de la paralysie générale des aliénés, p. 409.

107. Battarel. "Haematoma de la dure-mère." Journ. de Med. d'Algerie, VII., 266.

108. Janeway. "Peripachymeningitis," Boston Med. and Surg. Journ., XCVIII.

1880.

109. Mendel. Die Progressive Paralyse der Irren, p. 35.

110. Ledoux. "Hématome de la dure-mère." Bull. Soc. Anat. de Paris, LX., 64. Also Prog. Med. Paris, VIII., 668.

1881.

111. Peabody. "Pachymeningitis chronica." Med. Rec. N. Y., XX., 746.

1882.

112. Tuczek. "Zur Lehre vom Durhaematom," Irrenfreund.

113. König. "Ueber Pachy. haem. int." Berlin.

1883.

114. Duplaix. "Kyste hématique," L'Encéphale, p.522

115. Wernicke. Lehrbuch der Gehirnkrankheiten, Bd. III., p. 483.

116. Arndt. Lehrbuch der Psycheatrie, p.341

117. Deligny. "Contrib. a l'étude des osteophytes de la dure-mère dans la pachymén." Paris.

1884.

118. G. H. Savage. "Case of general paralysis with pachymeningitis." Journ. Ment. Sc., p. 261.

119. Camuset. "Note sur les lésions de la dure-mère cranienne dans la paralysis générale." Ann. Méd. Psycolog. II, 398.

120. Dixon. N. Y. Med. Rec., XXVI., 670.

121. Turner. Trans. Path. Soc. Lond., XXXVI., 16.

122. Savage and Wood. Journ. Ment. Sc., p. 261.

123. Totherick. Lancet, II., 106.

124. Mabille. Ann. Med. Psycolog., XII., 51.

1885.

125. Christian. "Pachyméningite avec symptomes de paralysie générale." Ann. Med. Psycholog., I., 233.

1886.

126. W. J. Mickle. General paralysis of the Insane, p. 278.

127. G. H. Savage. "Case of internal haemorrhagic pachymeningitis." Journ. Ment. Sc., p. 501.

128. Ord. Lancet, II., 1222.

1887.

129. J. A. Ormerod. "Subdural haematoma." Trans. Path. Soc. Lond., XXXVIII., p. 13.

130. Sainsbury. "Haematoma of the dura mater." Do. p. 12.

131. T. D. Savill. "Meningeal haemorrhage." Do.p.9

132. W. Hale White. "Old Meningeal haemorrhage," Do. p. 13.

133. Dana. Journ. of Nerv. and Ment. Dis. N. Y., XIV., 266.

1888.

134. Wiglesworth. "On haemorrhages and false membranes within the cerebral subdural space, occurring in the insane." Journ. Ment. Sc.

1889.

135. Dercum and Morey. University Med. Mag. Philadelphia, II., 509.

136. "Discussion on fibrinous membranes within the spinal canal in general paralysis." Brit. Med. Journ., II., 644.

137. Bevan Lewis. A Text-Book of Mental Diseases, p. 434.

1890.

138. Ziegler. "Pathologische Anatomie," Band II., Par. 129, page 373.

139. T. St. John Bullen. "Abstract of 1565 post mortem examinations of the brain." Journ. Ment. Sc.

140. J. W. Plaxton. "Case of chronic meningitis." Journ. Ment. Sc.

141. Obersteiner. The Anatomy of the Central Nervous Organs. Translated by Hill. p. 385.

1891.

142. E. B. Whitcombe. "case of pachymeningitis haemorrhagica interna following sunstroke." Journ. Ment. Sc., p. 557.

1892.

143. Wiglesworth. "Remarks on the pathology of so-called pachymeningitis interna haemorrhagica." Brain, p. 431. "Pachymeningitis interna haemorrhagica." Dict. Psycholog. Med., II., 877.

144. Goodall. "Note upon haematoma of the dura mater." Journ. Ment. Sc.

145. F. C. Hoyte. "pachymeningitis haemor. Int. chron." Med. Record, 30th April.

146. Clouston. Mental Diseases, p. 394.

147. Batty Tuke and Woodhead. Article "Pathology" in Diet. Psycholog. Med., II., 900.

148. Bauduy. Diseases of the Nervous System, p.148.

1893.

149. G. M. Robertson. "The formation of subdural membranes." Journ. Ment. Sc.,April and July.

150. H. C. Bristowe. "Two cases of pachymeningitis haemorrhagica interna." Journ. Ment. Sc., October.

151. Bondurant. "Pachymeningitis haemorrhagica interna, with report of eight cases." Alienist and Neurologist, XIV., 1.

152. L. Hirt. Diseases of the Nervous System. Trans. by Hoch., p. 5.

1894.

153. G.A.Sutherland. "On haematoma of the dura

mater associated with scurvy in children." Brain; (spring).

154. D. J. Hamilton. Text-Book of Pathology, II., 604.

155. Dagonet. Traité des maladies mentales. p.185.

1895.

156. Middlemass and Robertson. "Morbid conditions of the dura mater," (in mental diseases). Edinburgh Medical Journal, February.

REVIEW OF LITERATURE UPON SUBDURAL MEMBRANES.

Under the term "Subdural Membrane" I include all those morbid conditions which have been described by different authorities under the various names of "haemorrhagic pachymeningitis" (Pirotais, 48), "pachymeningitis haemorrhagica interna" (König, 113), "haematoma duraematris" (Virchow, 33), "meningeal haemorrhage" (Baillarger, 16), "arachnoid cysts" (Quain, 31; Wilks, 59;), "false membranes of the arachnoid" (Aubanal, 20), "meningeal apoplexy" (Prus, 23), "blood cyst of the dura mater" (Bauchet, 26), "subdural haematoma" (Omerod, 129), and "false membranes under the dura mater" (Clouston, 100). "Subdural membrane" is the term which has been adopted by G.M.Robertson (149), and it has the great advantage that, unlike most of the others, it may be used without implying belief in any particular etiological theory. As is evident from the foregoing list of literature the subject is one which has given rise to much discussion. It seems scarcely necessary here to give a sketch of its history, so frequently and so fully has this been done by previous writers, among whom may be specially mentioned Huguenin (92) and G.M.Robertson (149), and I shall confine myself to a statement,- as brief as

is consistent with the attainment of some degree of completeness,- of the different theories which have been held as to the morbid process at work in the production of subdural membranes, and of the various opinions which have been expressed, and the observations which have been recorded, regarding their occurrence and naked-eye and microscopic anatomy. I shall omit in this section all reference to the recent paper by Dr Middlemass and myself, to which I have already referred in the preliminary note, and my position in regard to which I have explained.

The majority of writers have endeavoured to explain all the different phenomena to be observed by one or other of two theories. Around these almost the whole of the controversy to which the occurrence of subdural membranes has given rise has waged. They are, first, the inflammatory theory, which is usually associated with the name of Virchow (33), and, second, the primary haemorrhagic theory, which has been specially advocated by Huguenin (92). The supporters of the inflammatory theory maintain that the process begins as an exudation upon the inner surface of the dura of a layer of fibrinous lymph which becomes organised, the false membrane increasing subsequently both by the deposition of fresh layers of the same kind, and by the occurrence of haemorrhages from the new vessels which develop in

it. The advocates of the haemorrhagic theory, on the other hand, maintain that inflammation plays no part in the process, and that all the phenomena may be explained as the result of an effusion of blood into the subdural space. Several authorities argued for the inflammatory theory before Virchow wrote, including Calmeil (7), Bayle (8) and Hesehl (28), and it has since been supported by Charcot (4), Lancereaux (43), Christian (52 and 81), Kremiansky (63), Rindfleisch (76), Camuset (119), Ziegler (138) and others. Prescott Hewitt (22) was the chief early authority who supported the haemorrhagic theory. Virchow's views were pretty generally accepted for some time after his paper appeared in 1856, but in 1877 Huguenin's contribution to the controversy, which is undoubtedly one of the most important which has appeared, gave a fresh impetus to the theory of primary haemorrhage. Wilks (54, 59 and 64), Sperling (71 and 72) and others, however, had previously combated Virchow's teaching, the two authorities named supporting their opinions by experiments upon animals. They injected blood into the subdural space and found that a typical membrane was formed. At the present day Virchow's theory seems still to be pretty generally accepted on the Continent, though with many important exceptions. In this country the majority of authorities, including

Greenfield (93), Wiglesworth (134 and 143) and Bevan Lewis (137), support the primary haemorrhagic theory. Other supporters of the latter are Baillarger (15 and 16), Dagonet (155), Brunet (37), Laborde (53,79 and 91), Prus (23), Lelut (14), Aubanal (20), Boudet (17), Rilliet and Barthez (19), Legendie(18), König (113), Crichton Browne (83) and Bondurant (151.) The advocates of the inflammatory theory maintain that their view is fully supported by the naked-eye and microscopic characters of the typical lesions which may be observed, and that the haemorrhages which are so frequently found occur subsequently to the formation of a membrane from the vessels of which the blood has escaped. Very numerous arguments against the inflammatory theory and in support of the haemorrhagic have been adduced, especially by Huguenin(92), Bevan Lewis (137), and Wiglesworth (134 and 143). Bevan Lewis urges against it the following considerations. (1) The cyst is readily removable from the dura. It is only slightly adherent or not at all. (2) In the majority of cases there is no evidence whatever of the existence of pachymeningitis; (a) the dura is not thickened or softened or vascular; (b) no organic connection exists between the two. (3) In early stages the characters are purely those of a simple extravasation of blood into the arachnoid cavity (subdural space). (4) There is the

co-existence in this affection of a recognised vascular disease and vasomotor disturbances which render haemorrhage frequent, e.g., the othaematoma or "insane ear." Wiglesworth also makes out a very strong case against the inflammatory theory. He states (134) that in one-sixth of the large number of cases of the disease which he has observed there was fluid blood, or this combined with recent clot, without the presence of any trace of membranes on the inner surface of the dura; that the membrane resembles in microscopic structure an organising thrombus; that the dura mater shows no signs of inflammation; and that the membrane can almost always be stripped off from the dura with ease, leaving the inner surface smooth and shining. In another paper (143, Brain) he says,- "If the membrane were the primary thing and the haemorrhage secondary, although we might get the membrane without the blood, we could not get the blood without the membrane," which condition, however, he says he has frequently observed. Others have pointed out that the so-called inflammatory membrane is often really a dense coagulum formed at the periphery of a blood-clot. Thus Baillarger (15 and 16) maintains that a delicate and transparent false membrane forms on the superior surface of the haemorrhage and another similar one on the inferior surface, and that these two unite at the circumference

into one layer which is often prolonged for a considerable distance. It should be mentioned that Obersteiner (141) holds that subdural membranes may develop either from a primary haemorrhage or as a result of inflammation of the dura. In 1877 Clouston (100) maintained that neither of the two theories satisfactorily explained the formation of false membranes under the dura in the insane. A new theory has recently been advanced by G.M.Robertson (149) in a paper of much importance, In his own words it is as follows;- "We believe that the all-important element in the production of subdural membrane formation is sudden lowering of the intracranial pressure, and that the effect of this is analogous to a dry-cupping of the dura mater." He holds that this sudden lowering of pressure arises from vaso-motor derangements in the cerebrum which cause vascular spasms and contraction of the brain. These vascular derangements, he believes, occur specially in certain forms of mental disease, notably general paralysis and senile insanity. He maintains that the sudden lowering of pressure induces distension of the superficial vessels of the dura and numerous minute haemorrhages from them. His theory is thus one to account for the primary haemorrhage, rather than one which is opposed to both of the older views. He adduces in support of his contentions

very numerous and elaborate arguments, drawn partly from phenomena which he has observed, partly from considerations which are more or less theoretical, and also from actual experiments which he has made.

Regarding the later stages of the morbid process the various authorities are fairly agreed. The inflammatory exudation or the blood-clot becomes replaced by granulation tissue, the vessels of which (derived always from the dura) are extremely liable to rupture. In this way new blood clots are formed either within the membrane, below it or on its surface, and lead to the development of more granulation tissue. Thus the membrane increases in thickness, though, as already stated, the advocates of the inflammatory theory maintain that it also grows as the result of the exudation of new layers of inflammatory lymph from the dura.

Very careful descriptions of the naked-eye appearances of subdural membranes have been given by various writers, among whom Wiglesworth (134) specially deserves mention. According to the advocates of the inflammatory theory "the first morbid sign is the appearance of very thin fibrinous deposits on the internal surface." (Ziegler, 138). Those who hold the haemorrhagic theory maintain that this is either a white, or a decolourised, blood-clot, and that the earliest stage is really represented by a

layer of freshly coagulated blood. The occurrence of rusty staining on the inner surface of the dura without the presence of a separable membrane seems scarcely to have been noticed until G.M.Robertson (149) specially drew attention to it, and claimed that it represented an important stage in the pathological process in many cases. Descriptions of some of the appearances presented in more advanced cases, or at least in those in which there are larger membranes, may be quoted from Wiglesworth (134). "A thick gelatinous rather firm membrane." "A thin whitish-pink gelatinous more or less coherent lamina;" "A thick organised fibrinous layer." "A coherent membrane spread over the inner surface of the dura." "A thin non-coherent gelatinous lamina, dotted over at places with punctiform haemorrhages." It may be added, that the membrane is almost always of a soft gelatinous character, and that when organised it appears usually highly vascular. It may be a very thin layer, or attain to half an inch in thickness, or even more. It may be distinctly laminated. It frequently presents small, more rarely large, recent haemorrhages into its substance. It may have a rusty tinge owing to the presence of haematoidin granules. Bevan Lewis (137) states that in old membranes calcareous matter may occasionally be found. In some cases the membrane is formed of two

layers which enclose fluid. This is the cystic form. It assumes various appearances. It may be localised to a small area or extend over the whole of one side of the brain, and even may be developed on both sides. The walls of the cyst may be thin or thick. The contents may be recently effused blood, disintegrating blood, or, rarely, almost clear fluid. These cysts are generally explained as a late result of either a large primary haemorrhage, or of a large secondary haemorrhage between a false membrane and the dura. (Bauchet, 26). The membranes are always adherent to the dura, though they are as a rule easily detached. They are almost never adherent to the arachnoid. They are usually uni-lateral, but are also frequently bilateral. About these points there is general agreement.

Regarding the source of a primary haemorrhage into the subdural space, Crichton Browne (83), Huguenin (92), Bevan Lewis (137) and Wiglesworth (134 and 143) are all agreed that it is usually to be found in a pial vein. G.M.Robertson (149) on the other hand holds that the haemorrhage frequently occurs from the superficial dural capillaries.

Subdural membranes are most frequent over the upper and lateral surfaces of the brain. Still many cases have been described in which they were confined to the basal fossae. Bevan Lewis and

Wiglesworth both say that they have never seen either recent clots or organised membranes in the cerebellar fossa.

Various explanations have been offered to account for the frequency of haemorrhage from the new vessels in a membrane. Pirotais (48) states that it is owing to the fact that they have a great tendency to fatty degeneration. Lancereaux (43) holds the same opinion. Rindfleisch (76) gives another explanation. He says that in the normal capillaries of the dura which lie in dense tissue, the blood pressure is excessive, while the new capillaries of the membrane which communicate with them are in a soft tissue which cannot withstand the pressure which is transmitted to them. Hence they dilate and rupture.

All authorities are agreed that subdural membranes are much more common in the insane than in the sane. Regarding their occurrence in the insane the following statistics may be quoted. Wiglesworth (143) observed 54 cases in 637 post mortems. (8.47 per cent). Crichton Browne (83), found them in 5 per cent of cases. Bevan Lewis (137) found them in 81 cases out of 1565, or in 5.2 per cent. Bullen (139) found them in 7.6 per cent of cases. They occur in the insane most commonly at a somewhat advanced age. According to Wiglesworth the average

is 51.6 years. Bullen, however, whose authority is of special value on account of the large number of records upon which his statistics are based, found that they were most common between the ages of 40 and 50. It is agreed that they occur in the great majority of instances in patients in whom the mental disease is of long standing. The form of mental disease which furnishes the largest proportion of cases is general paralysis. Wiglesworth found that exactly half of the subdural membranes that came under his notice occurred in this disease; and the statistics of Crichton Browne and Bevan Lewis bring out a similar result. Taking general paralysis alone, Bullen found subdural membranes in 13.4 per cent of cases, Bevan Lewis in 12 per cent and Christian (81) in as many as 30 per cent. Regarding the occurrence of subdural membranes in the mentally sound, according to Althaus (94) they occur "in the old, decrepit, worn-out, intemperate," and are "not unfrequently associated with low forms of pleuro-pneumonia, pneumothorax, pleurisy, pericarditis, emphysema, rheumatic fever, delirium tremens, leucaemia, pernicious anaemia, scurvy, haemophilia and various forms of Bright's disease of the kidneys." Many other writers speak of having observed cases in these or in other bodily diseases, among which should be specially mentioned typhus fever and those associated with chronic alcoholism. G.A.Sutherland

(153) has specially directed attention to the occurrence of haematoma of the dura mater in infantile scurvy. As evidence of the degree of frequency of the occurrence of subdural membranes in the mentally sound it may be mentioned that Kremiansky (63) obtained the material for his authoritative work entirely from patients who died in a general hospital. It is generally admitted, however, that except in drunkards, and excluding cases which are manifestly due to the recent rupture of a vessel, the condition is rare in the mentally sound.

Many authorities have endeavoured to give a satisfactory explanation of the special frequency of subdural membranes in the insane. Wiglesworth(143) thinks it is due to "wasting of the hemispheres and general or localised congestion of the meninges." He believes that the haemorrhage is often simply compensatory. Crichton Browne (83) and Huguenin (92) hold practically the same view. G.M.Robertson's explanation (149) has already been given.

Regarding the microscopic anatomy of subdural membranes remarkably little has been written, considering the mass of literature which now exists about them, and still less has been recorded as to associated microscopic changes in the dura. The various descriptions which have been given of the minute anatomy of the membranes may be summed up as

implying that they may be composed of red or white blood clot, or of fibrin threads entangling numerous leucocytes (inflammatory exudation), or of granulation tissue. When of the last named structure they usually contain very numerous large thin-walled, often varicose capillaries, and more or less abundant haematoidin granules. Greenfield (93) observed on the free surface of the membrane a layer of endothelial cells. König (113) states that many of the cellular elements in organising membranes are "due to proliferation of the epithelium of the dura." Fatty charges in the vessels have been observed by Pirotais (48) Lancereaux (43) and others. Congestion of the dural vessels is mentioned by many writers as occurring whenever there is a membrane, and those who believe in the inflammatory theory maintain that there is in addition always a small-celled infiltration of the superficial dural tissues. G.M.Robertson (149) has drawn attention to the frequent occurence of dilated vessels and deposits of haematoidin granules near the inner surface of the dura in cases in which there is no membrane. The same writer has described the frequent occurrence of very delicate fibrinous films on the inner surface of the dura in association with congestion. Obersteiner (141) states that "not rarely, in old people

especially, concentrically laminated glancing concretions, corpora arenacea, are found in the dura." He says they consist of phosphate and carbonate of lime. He also mentions that they may be found in subdural membranes.

NOTE ON THE STRUCTURE AND FUNCTIONS OF THE DURA MATER.

There are certain facts regarding the microscopic structure of the dura mater to which it is necessary for me to draw attention, in order to render intelligible the succeeding descriptions of morbid appearances observed in it. Many of these facts, though of the utmost importance in relation to subdural membrane formation, seem to have been unknown to previous writers on this subject, almost without exception. Some of them have already been drawn attention to in the recent paper published along with Dr Middlemass, (156), but I have now some additional observations to make. I shall refer to some minor points first.

Many writers in describing the microscopic characters of cerebral subdural membranes mention the elastic fibres of the dura. According to Sappey, however, elastic fibres exist only in the spinal dura From my own observations I can corroborate this statement, and I would add to it that they occur there in very large numbers, - a fact which may have some bearing upon the question of one of the functions of the dura. Some of the microscopic

specimens produced to show morbid conditions will serve at the same time to demonstrate these points. Specimens No. 60 and 81 are transverse sections of the cerebral dura, while No.108 is a similar preparation from the cervical region. In the last, which is a haematoxylin and eosine preparation, the elastic fibres are seen deeply stained with eosine. These elastic fibres are also very prominent objects in horizontal sections of the spinal dura. They are seen in specimen No.127.

Another point of minor importance is the layering of the dura. The membrane is usually described as composed of two layers of dense fibrous tissue running in different directions, and some authorities even mention a definite relationship between the thickness of each. Thus Alexis Thomson ("Pachymeningites Chronica Externa," Journal of Pathology and Bacteriology, October 1893.) says, "The bundles of fibrous connective tissue of which it is composed are arranged in two layers: of these the inner is only one-sixth of the thickness of the outer, while it is denser in the arrangement of its fibres." If, however, some of the transverse sections produced for other purposes are examined, they will serve to show that these statements are inaccurate. The most frequent arrangement, and the constant one near the venous sinuses, is no doubt that of two layers

(Fig.1), but in other situations all the fibres of the membrane are frequently seen to run in one direction, and to form only one layer, while again, sometimes three distinct layers may be seen. When two layers exist there relative thickness varies greatly in different instances. In all probability the arrangement is constant for each definite area of the dura, but this is a subject which I have not worked out. The following specimens will serve to demonstrate the above points. Showing one layer, Nos.85, and 31. Showing two layers, Nos.1, 60, 81, 93. Showing three layers, No.126.

The characters of the normal connective tissue corpuscles of the dura should be recognised, as in some morbid conditions connected with subdural membrane formation they can be shown to be altered in appearance. With haematoxylin and eosine staining, their nucleus is a small deeply stained elongated spindle. The protoplasm cannot, as a rule, be distinguished. These cells are seen in specimen No.1.

The next point is one of great importance in relation to subdural membrane formation, namely, the normal vascular arrangements of the dura. The statements which I wish to make regarding them are in the main the same as those which were made in the recent paper by Dr Middlemass and myself, (156), and from it I shall quote. "Only one writer upon this

subject(Obersteiner (141)), as far as we have been able to ascertain seems to be aware of the fact that these arrangements are, in one particular, of a highly specialised character, though he attaches no importance to it in the above connection. While the main arteries are placed towards the outer surface, the large veins are found near the centre between the two layers, when these exist. The greater part of the thickness of the dura is only scantily supplied with capillaries. Near the inner surface, however, there is an exceedingly rich capillary network, the minute structure and relations of which can be satisfactorily studied only in horizontal sections The first point that attracts attention about these vessels is their size. We are satisfied that, even in normal conditions, they are three or four times the diameter of an ordinary capillary. Their calibre is always uniform, and they show no localised dilatations in the healthy state. Many of them run in little grooves close to the surface, covered only by endothelium, (Fig.2). The venous radicles into which the capillaries pass have exceedingly thin walls, and are relatively of very large size. When the capillaries are examined in horizontal sections of the dura a narrow clear space appears to lie on each side of them. These are the perivascular canals of the dura to which we wish to direct special

attention. (Figs. 2 and 3). They are mentioned by Obersteiner as occurring round the arteries only. We have studied them very carefully as they occur in the human subject, and we find that they constantly accompany the arterioles and capillaries throughout the dura, but not the veins. As far as the arterioles at least are concerned, they are probably to be regarded as a special development of the normal perivascular lymph-channels. Though the appearance round the capillaries, when examined in horizontal sections, is that of a double canal, it can yet be easily made out that the arrangement is really that of a single channel in the centre of which the capillary is suspended by delicate strands of fibrous tissue here and there, which pass obliquely across the space. A layer of endothelium lines the outer wall of the canal. Its inner wall is, we believe, that of the capillary. We have been unable to detect any special endothelial layer covering the outer surface of the capillary. In transverse sections it is almost impossible to distinguish these perivascular canals, probably owing to the circumstance that in such preparations they tend to collapse. Obersteiner states that they communicate with the subdural space by means of stigmata on the visceral surface of the dura, and that on the other hand, "they open into the real blood-vascular system." The

latter statement is, we think, almost certainly an error. We have likewise been unable to see any of the transverse connecting branches of these canals which are described by him."

In specimen No.2, which is a superficial horizontal section, the normal capillaries and perivascular canals may be seen at the spot marked +— In specimen No.3 at the spot marked +— an artery, capillary and vein communicating with each other may be observed. Superficial capillaries may be seen in transverse section in Specimen No.1 at the spot marked +— . (Fig.1). In specimen No.4 at the spot marked +— there are seen large ampullary dilatations of the superficial capillaries. (Fig.4). G. M.Robertson (149), looks upon these as a morbid manifestation, but so frequently have I observed them in apparently otherwise healthy duras, that I am inclined to regard them as normal features. The opening of the perivascular canals upon the surface will be referred to again.

The next question is also one of considerable importance in relation to subdural membrane formation. It is the structure of the inner surface. No one now believes in the existence of a parietal arachnoid, but there are still many writers who speak of a subendothelial layer of loose connective tissue on the inner surface of the dura. I have already

(156) committed myself to the view that the endothelium in its normal condition lies immediately upon the dense fibrous tissue of the dura. Now, while this view is certainly borne out by all the normal transverse sections I have examined (See for example specimen No.1), I have recently observed a number of appearances in superficial horizontal sections, which I can only reconcile with the existence of a very delicate subendothelial layer of loose connective tissue fibres. It is only very occasionally that this appearance presents itself and, therefore, it is possible that the arrangement only exists in certain parts. If the connective tissue fibres in question were parallel with those of the deeper tissues, the appearance might be regarded as a fallacious one, but they present quite an irregular arrangement so that one is bound to admit that they constitute a separate layer. There, of course, remains the possibility that such a layer is a pathological formation. The appearance referred to is seen in the following specimens. No.5 at the spot marked ⊢ . No.190 at the spot marked ⊢⊢ . No. 192 at the spot marked ⊢⊢ . The characters of the normal surface endothelial cells (which form only a single layer) may be seen in specimen No.5. Their cell-plate can seldom be seen in haematoxylin and eosine preparations. Their nucleus is a large

oval structure which stains somewhat faintly with haematoxylin. In its centre there is a more deeply stained large oval nuclear network, part of which, no doubt, forms a nucleolus. This central portion with ordinary powers usually appears as if composed of minute, closely set, dark dots. One normal nucleus is shown in fig.30, and the extent of the cell-plate is indicated in fig.31. The nuclei of the endothelial cells of the perivascular canal walls have the same appearance. The cell-plates may be seen in specimen No.98, which is a superficial horizontal section of a silver preparation. This specimen also serves to demonstrate the way in which the perivascular canals probably open upon the surface of the dura. As already stated, Obersteiner believes that they communicate with the subdural space by means of stigmata. Such structures certainly cannot be distinctly seen either in this specimen or in many others of a similar kind, which I have examined. The arrangement that appears to exist is that over many of the superficial perivascular canals, but especially over certain wide expansions of them, the surface endothelium stops opposite the edge of the canal, which is bridged over solely by its own endothelium. I have been unable to observe any distinct openings, and if fluid passes from these perivascular canals into the subdural space, - as I have

little doubt it does, - it is probably merely by a temporary parting asunder of some of these cells.

The last point which I wish to refer to regarding the normal structure is one which is of importance, because of its bearings upon one of the many difficult problems raised by the microscopic appearances to be observed in superficial horizontal sections of the morbid dura. On either side of the superior longitudinal sinus there are seen quite distinctly with the unaided eye long fibrous tissue buttresses extending outwards and obliquely forwards or backwards over the inner surface of the dura. They may be seen with the unaided eye, as well as with the microscope in specimen No.156. Similar, but much smaller , dense fibrous tissue strands occasionly occur in other parts of the dura. One is seen in microscopic specimen No.6, at the spot marked +—. As will be shown further on, such strands are often practically indistinguishable from certain structures, which probably develop as the result of a morbid process.

What I have to state here regarding the functions of the dura has already been published in the recent paper referred to, and I shall again quote from it. "The functions of the dura are usually stated to be two in number. First, it is an internal periosteum for the skull, and second, it

serves to protect and support the brain and its vessels. It has, however, a third function, to which attention is specially directed by Foster (Text book of Physiology, 1890, p.1133.) - namely, the regulation of the intracranial pressure when its normal equilibrium is disturbed by the expansion and contraction of the brain, which attends its physiological activity. He believes that it subserves this function by means of its venous sinuses. "Within the limits of the normal cerebral circulation, the characteristic venous sinuses especially serve to regulate the internal pressure; they form temporary reservoirs from which a large quantity of blood can be rapidly discharged from the cranium." (Loc. cit.) We think there can be no doubt that this important function is in part performed by these sinuses. Not only will blood be forced out of them when the brain expands, but an opposite condition of distension will be produced in them when the brain diminishes in volume. At the same time the mechanism by which the intracranial pressure is regulated is undoubtedly a much more complex one than this, and has other important factors, as Foster himself points out. Some room, he says, may be provided for an extra quantity of blood in the brain by an escape of cerebro-spinal fluid. In this relation it is important to bear in mind that it has

been experimentally demonstrated by Naunyn and Schreiber (Arch. f.exper.Path. u. Pharmacol., XIV., 1881, p.1), that a free communication exists between the cushion of liquid which envelops the brain, and that which surrounds the spinal cord. Now, while it is mainly in these two ways that the normal intra-cranial pressure may be preserved, when an increased flow of blood to the brain causes it to expand, it yet seems to us improbable that, in the case of a cerebro-spinal axis, the usual position of which is vertical, a return of cerebro-spinal fluid into the cranium cavity can be an important factor in the maintenance of the normal pressure when the brain contracts. The authors just quoted found that pressure applied to the contents of the sub-arachnoid space of the cord was not transmitted so rapidly to the cranial cavity as pressure applied in the opposite direction. It is more probable that when the brain contracts the normal pressure is preserved by a distension of the dural sinuses and an increased secretion of cerebro-spinal fluid. Since contraction of the brain means diminished blood-supply, the extra quantity of cerebro-spinal fluid required can scarcely be supplied from its usual source in the choroid plexuses. It is generally stated that there is normally only a small amount of fluid in the sub-dural space. It is certain, however, that the

fluid which it does contain is secreted from the dural vessels, and that it passes in a constant, though doubtless small, stream through the space, finding exit by special channels provided in the walls of both the spinal and cranial cavities. Pathological states prove that it is capable of being secreted in large quantity, and we are inclined to think that, under normal conditions, it is secreted in much greater amount than is generally believed. We, indeed, would suggest that the regulation of the intracranial pressure is, in part, a function of the whole dura mater, and not merely of its venous sinuses, and that, when the vessels of the brain contract, one of the factors in the mechanism by which the normal intracranial pressure is maintained, though it may only be a subsidiary one, is an increase of the normal secretion from the vessels of the dura into the subdural space. The peculiar perivascular canals, by means of which, it is evident, fluid may be drawn from a large vascular area and poured out upon the dural surface, are, we think, a special adaptation to this function."

I would emphasize the fact that the above is offered merely as a <u>theory</u> which may possibly help to explain a very difficult pathological problem. The matter will be referred to again.

DESCRIPTION OF THE NAKED-EYE AND MICROSCOPIC APPEARANCES PRESENTED BY THE DURA MATER IN A SERIES OF ASYLUM AND GENERAL HOSPITAL CASES.

CASE No. 1.

J. C., male, aged 25.

Case of advanced general paralysis. Patient was "in a series of congestive attacks" for ten days before death. He had frequent previous congestive attacks.

Post mortem 30. 8. 94. Typical morbid appearances of general paralysis found in brain.

Naked-eye appearances. Dura appeared normal except that there were some granulations over the sphenoid. There was no membrane or rusty staining.

Microscopic appearances. In superficial horizontal sections from over sphenoid and from vault very marked vascular and other morbid changes can be seen, though at the same time there are extensive areas which appear quite healthy. In most of these sections,- all of which it is to be understood give a view of the inner surface of the dura,- there are numerous solid hyaline rods which in alum carmine and eosine preparations stain deeply with eosine, and in haematoxylin and eosine specimens stain either with eosine alone or with both dyes. They are of about the diameter of the normal superficial capillaries of the dura, and have usually a slightly striated appearance under high powers. A great

variety of intermediate forms can be seen between these hyaline rods and apparently healthy vessels.

The perfectly hyaline rods may be seen in micro. spec. No 7 in many places. They stain deeply with eosine. (Fig. 36) Some of the intermediate forms are also to be observed in this preparation. The hyaline rods can occasionally be traced into patent capillaries. The spot marked ++ shows a thickened and occluded vessel with some granules of altered blood-pigment within it. (Fig. 59). The walls of some of the vessels show only slight hyaline thickening. (Micro. spec. No.8 Fig. 16). In others in addition to the thickening there is marked aggregation of cells upon them or in their peri-vascular canals. (Fig. 15). See alum carmine preparations. The diminished lumen of these vessels may contain some blood corpuscles or it may be empty. The hyaline rods frequently show a small aggregation of cells upon them. (Micro. spec. No. 9 + Fig. 38.). Similar cellular aggregations may occasionally be seen on apparently healthy vessels. In other cases the wall of the perivascular canal seems to become affected by hyaline thickening, while the capillary wall remains normal. (Micro. spec. 9 ++ Fig. 18). The wall of both the capillary and the perivascular canal may also be affected. (Micro. spec. 7 +++). There is much altered blood-pigment lying outside the vessels

in some parts. It is sometimes in the perivascular canal. (Fig. 19). These pigment granules may be lying near degenerated vessels, or outside of dilated and very thin-walled capillaries which in some preparations are very numerous near the surface, often forming networks. (Fig. 5) See Micro. spec. 9, 10, 11, 12. In haematoxylin and eosine preparations thin irregular sheets or flattened networks of a slightly granular and sometimes fibrillated substance which contains some cellular elements and stains with both dyes, may be seen on the surface. (Micro. spec No 13). They are continuous with many of the hyaline rods. (Micro. spec. 7 Fig. 54). It can occasionally be seen that there is a continuity between them and the wall of a perivascular canal. This is shown in specimen 10 at the spot marked +— , (Fig. 57). It may often be observed that the hyaline rods, as well as many of the intermediate forms, are superficial to the granular sheets. See specimen No 7 at spot marked ~~+~~.

Concentric bodies are numerous in some of the preparations. (Micro. spec. No 14 Fig. 49). Thin-walled capillaries may sometimes be seen in close relation to them. Seeming transition forms between them and the hyaline rods may be observed. Micro. spec. ~~9~~ 11 and 14. Figs 40, 42, 41).

In the alum carmine preparations some small

patches may be seen near the surface which stain more deeply than the rest of the tissues and are faintly granular. They are not numerous.

Granulations on the sphenoidal dura appear as dense aggregations of somewhat large cells. Spec. 14 ## In the same preparations there are also areas over which there is a slight general increase in the cellular elements on or near the surface.

Sections stained with methyl violet show no waxy reaction.

Transverse sections of a piece of hardened dura from the base which showed three small dark spots contain peculiar degenerated areas. Two such areas of large size are seen in the specimens but other much smaller ones of a similar character can also be observed. The larger areas lie just below the inner surface. They are somewhat oval in shape and in breadth are equal to about half the diameter of the dura. They show most distinctly stained with haematoxylin and eosine. (Micro. spec. No. 15 Fig. 61). Three or four different zones may be distinguished, but as these are rather irregular in form they have a somewhat different relationship to each other in different specimens. In haematoxylin and eosine preparations the outer zone is somewhat deeply stained of a purple colour. It is usually narrow and has a fibrillated and slightly granular

appearance. One or two deeply stained nuclei may be seen in it. In some of the preparations it is joined at one side with a large central area of similar characters. In this the outlines of degenerated granular connective tissue cells can be made out faintly stained of a purple colour. Deeply stained nuclei occasionally occur. Immediately external to this central area there is a hyaline zone devoid of cells. It stains very deeply with eosine. Between this and the outermost zone there is at places an area of tissue closely resembling the normal dura in structure but staining more faintly. In some specimens the central zone is entirely cut off from the outermost. These areas show no waxy reaction with methyl violet. Osmic acid preparations show some small black granules in the degenerated cells of the central area.

In transverse sections from the vault (Micro. spec. 16 & 17) the thickening of the capillaries or perivascular canal-walls can be made out at places. Some spots of altered blood-pigment can also be seen. There are some areas near the inner surface which show a slight increase in the cellular elements. Several concentric bodies may be seen cut in transverse section. The one shown in specimen 17 is distinctly covered by the dural tissues and is not merely attached to the surface.

CASE No. 2.

H. P., male, aged 35.

Case of advanced general paralysis. Patient had a congestive attack a fortnight before death. He rallied for a time and sank gradually.

Post mortem 15.3.94. Typical morbid appearances of general paralysis were found in the brain.

Naked-eye appearances. There was a false membrane over almost the entire inner surface of the dura. It was delicate except over the left anterior fossa where it was about one-eight of an inch in thickness, tough and dark red in colour. Elsewhere there was marked rusty staining. There were especially at the vertex numerous minute haemorrhages.

Microscopic appearances. The false membrane where thin enough to be examined as a stripped preparation shows the structure of granulation tissue, or of recently vascularised blood-clot, with special features. (Micro. spec. 18 & 19). There are very numerous delicate capillaries, some of which are considerably dilated. Altered blood-pigment is in great abundance. In some of the intervascular areas there are numerous delicate fibrils which are probably the remains of the fibrin threads of a blood clot. (Micro. spec. 20). In the intervascular areas the cells, which are not specially numerous, except at

places in some preparations, are of several kinds. Mononucleated leucocytes are most common, some having a small quantity of surrounding protoplasm and others a large amount. (Fig. 33). Many of them contain altered blood-pigment. (Fig. 34.). There are also numerous large cells which appear of a pale blue colour in haematoxylin and eosine preparations. They have either no nucleus or a large somewhat faintly stained one. They frequently contain small lightly stained bodies,- an appearance which may, however, be due to vacuoles.(Fig 29) There are also present some cells with nuclei resembling those of the normal endothelial cells of the dural and arachnoid surfaces. There are also some small, deeply stained spindle shaped nuclei which probably represent fibroblasts. Some preparations show numerous spindle cells, small capillaries and a generally more dense structure, probably indicating an older and more highly developed tissue. (Micro. spec. No 21. Fig. 9). In some preparations there are fairly dense aggregations of cells, the exact character of which is doubtful. Concentric bodies are numerous in this membrane. (Micro. spec. 18, 19, 22). Osmic acid preparations show absence of any very marked fatty changes. Some fatty capillaries, however, may occasionally be seen. (Micro. spec. 23). Specimen 24 shows a delicate membrane stripped from the cerebral

surface of a fairly thick portion. It probably represents a membrane of more recent formation. It contains large areas without any vessels, and remains of fibrin threads can be distinctly made out. There are numerous red corpuscles and much altered blood-pigment, most of which is contained in leucocytes. The large pale cells already described are very abundant. There are also in large numbers endothelial cells of the kind already referred to. Fig. 26). Their nuclei show toward their centre numerous minute deeply stained dots which represent either a nuclear network or nucleolus. Division of this central structure into two or three portions may occasionally be observed. The surrounding protoplasm is usually difficult to see but in the case of some of the cells it can be easily distinguished.

Superficial horizontal sections of the dura below the false membrane show various morbid appearances. Over large areas there is on the dural surface a distinct layer of new tissue with very numerous capillaries, outside of which there is at places much altered blood-pigemnt. There are some small recent haemorrhages. (Micro. spec. 25). The cellular elements are numerous and consist of spindle cells, leucocytes and endothelial cells. Many of the leucocytes are filled with haematoidin

granules, while many of the endothelial cells show degenerative changes,- mainly vacuolation and swelling up into a pale faintly granular mass. (Fig. 27). Other parts show the original dural surface and its vessels. Many of these show morbid changes. Some show aggregation of cells around them,- probably chiefly endothelial. (Micro. spec. 26"). Others are markedly thickened, some being converted into solid rods. (Micro. spec. 26' and 27'). Others again show a large amount of altered blood-pigment lying outside their walls which often appear thickened and granular. (Micro. spec. 28 and 29). Numerous torn vessels which have passed to the false membrane may be seen. Some concentric bodies are present, and there are also some appearances which are probably transition forms of these structures.

Median horizontal sections show no very distinct morbid changes. (Micro. spec. 30).

In transverse sections of the dura with false membrane attached the structure of the latter as already described can be less distinctly made out than in the stripped specimens. (Micro. spec. 31 Fig. 7). The subjacent dura appears healthy. In some preparations, however, its vessels are much congested and dilated. Sections through what appeared to the naked eye as a minute haemorrhage show under the microscope a small aneurismal dilatation in the

false membrane. (Micro. spec. 32 , and 33 which shows the edge of the wall). In Micro. spec. 34 medullated nerve fibres are seen in horizontal section stained with osmic acid. They show slight varicosity, but it is doubtful if they are much degenerated. Micro. spec. 35 shows on the surface of the membrane some flattened bodies which blacken with osmic acid. Micro. spec. 36 is a transverse section through the thick portion of the false membrane already referred to. It shows a large haemorrhage into its centre, extending longitudinally.

Oblique sections of the dura with false membrane attached show the structure of the latter very well, and occasionally the vascular connections between the two can be traced. (Micro. spec. 37 and 38). The nuclei seen in the false membrane are mainly of the endothelial type,- pale and oval with a finely dotted oval nucleolus. A small amount of surrounding protoplasm faintly stained with eosine (haematoxylin and eosine staining) may occasionally be seen. These cells are especially numerous next the dura. Large pale blue vacuolated cells are also very plentiful. They present very varied appearances. Many of them are irregular glassy-looking bodies. Others are somewhat granular and show what look like vacuoles. (Fig. 28). There

are numerous extravasated red blood corpuscles, and also much altered blood-pigment both within leucocytes and lying between the cells. There are some slight aggregations of cells around the vessels of the dura which again are mainly of the endothelial type.

CASE No. 3.

E. S., female. Age 60.

Case of Senile Insanity. Patient sank gradually

Post mortem 7.8. 94. Extensive areas of softening found in cortex and in white matter.

Naked-eye appearances. The dura was markedly adherent to the calvarium. On the surface of the falx on the right side there was a small bony spicule about the size of, and the same shape as, a melon seed. The dura showed no thickening, membrane or rusty staining.

Microscopic appearances. In superficial horizontal sections of dura from vault some of the capillaries are surrounded by dense aggregations of cells. (Fig. 20). There are numerous small granular patches near the surface which stain deeply with eosine. (Micro. spec. 39+). Some concentric bodies are present. There are small collections of red blood corpuscles lying outside of some of the capillaries. (Micro. spec. 39++). In superficial horizontal sections stained with haematoxylin and eosine there are seen at places on the surface thin sheets and networks of a slightly granular material which stains with both dyes. It contains numerous oval nuclei. (Micro. spec. 40). Fig. 55.

CASE No. 4.

R. C., male. Age 60.

Case of Senile Insanity. Patient died from an attack of pneumonia.

Post mortem 6.9.94. Catarrhal pneumonia. Small cortical atrophic areas in cerebrum..

Naked-eye appearances. The dura was much congested. There were numerous small spots of quite recent haemorrhage on the inner surface. They were most numerous at the base. There were some present in the cerebellar fossa. The blood was in the form of dark clots with a very delicate colourless film extending from them. There were some small granulations over the sphenoid.

Microscopic appearances. Membrane formed by blood-clot, stripped from the dura after hardening in potassium bichromate, shows a network of fibrin threads entangling red and white blood corpuscles. There are also numerous endothelial cells distinguished by their large oval nuclei. (Micro. spec 41) In superficial horizontal sections many of the superficial capillaries are greatly distended with blood, some of them appearing varicose. (Micro. spec. 42) There are numerous small extravasations of blood around these distended vessels. (Micro. spec. 43 Fig. 22). Many of the capillaries show slight thickening and granularity of their walls, and some aggregation of cells upon them. (Micro. spec. 44).

Many of the thickened and granular capillaries contain no blood corpuscles. In micro. spec. 45 there is seen in a perivascular canal a collection of cells behind which the vessel is slightly dilated and shows escape of red corpuscles from its lumen. (Fig. 21). Some concentric bodies are present. Superficial horizontal sections treated with osmic acid show very occasionally an oval or rounded black mass in a vessel, often completely filling it. (Micro. spec. 46 Fig. 23). There are numerous round cells in and immediately outside of the vessel. Median horizontal sections show in some of them a recent haemorrhage burrowing longitudinally between the connective tissue fibres.

CASE No. 5.

J. S., male, aged 34.

Case of advanced general paralysis. Patient sank gradually. Did not die in a congestive attack. Post. mortem 6.12. 94. Numerous small pulmonary abscesses.

Naked-eye appearances. Slight adhesion of dura to calvarium. Small bony spicule in falx major. Marked general thickening of dura in neighbourhood of right middle meningeal artery. On the inner surface of the dura, extending over the whole of the right side, there was a very delicate gelatinous-looking false membrane. It was thickly dotted over with dark red or rusty points. With the aid of a hand lens some minute vessels could be seen in it. It was easily detached from the subjacent dura, the surface of which was gelatinous-looking and showed slight yellow or rusty staining. Much blood adhered to the outer surface of the dura. It seemed partly extravasated into the dural tissues. On the left side the dura appeared normal. There were some granulations over sphenoid.

Microscopic appearances. Stripped preparations of hardened false membrane show it to consist of granulation tissue with very numerous and much congested capillaries. There is copious altered blood-pigment

in the intervascular areas, and also numerous extravasated red corpuscles. (Micro. spec. 47 Fig. 8). Fibrin threads can be seen in some of the capillaries (Micro. spec. 48). There are some concentric bodies in the false membrane. Superficial horizontal sections of dura below false membrane show slight general increase in the cellular elements. Capillaries, which are very numerous, are in many cases greatly distended with blood. Many of them are evidently new vessels as they show no perivascular canals. (Micro. spec. 49-50). Numerous recent blood extravasations can be seen from them. (Fig. 12) The original capillaries and small arterioles are in many instances somewhat thickened and granular-looking, and studded with cells. Many of these vessels are empty, while others contain only a narrow string of corpuscles in an evidently diminished lumen. (Micro. spec. 50). There is much altered blood-pigment lying in the intervascular areas both in relation to the new capillaries and the original dural vessels. See preceding micro. spec., and also No. 51 which is a superficial horizontal section made with membrane in position, the outer or cut surface being turned towards the coverglass. A superficial horizontal section of dura under a recent clot, with this in situ, when examined from outer surface, shows greatly distended capillaries.

(Micro. spec. 52). Superficial horizontal sections stained with osmic acid show no fatty charges, (Micro. spec. 53). Some concentric bodies are seen in these preparations. Horizontal sections of dura near outer surface show greatly congested vessels and numerous recent capillary haemorrhages. There is also much altered blood-pigment in the tissues. (Micro. spec. 54) Some of the small vessels contain no blood. In one specimen there is seen a capillary containing some round cells but no red blood corpuscles, while behind this the vessel is greatly distended with blood and the wall ruptured.

Horizontal sections through dura near superior longitudinal sinus show numerous recent haemorrhages, but no altered blood-pigment. The sections pass through Pacchionian bodies in which some concentric bodies may be seen. (Micro. spec. 55).

In superficial horizontal sections of sphenoidal dura which showed granulations there are seen numerous much distended and often varicose capillaries with some recent haemorrhages (Fig. 13 Micro. spec. 56), altered blood-pigment outside of vessels, and on the surface aggregations of deeply stained cells, probably endothelial in character.

CASE No. 6.

A.S., male, aged 40.

Case of choreic insanity. Ill for 17 years. Died gradually from phthisis.

Post mortem 12.3.94.

Naked-eye appearances. The dura seemed quite normal.

Microscopic appearances. Superficial horizontal sections show morbid changes especially in the surface endothelium and the superficial vessels. There is also in places on the surface a thin granular and slightly reticulated film (Micro. spec. 57+). This occasionally appears as a somewhat hyaline substance of a deep purple colour in haematoxylin and eosine preparations, which tends to form net-works and to contract into curled masses. (Micro. spec. 57+ and 58). In the latter specimen at the spot marked a long rod of a more crimson tint is to be observed. It may be a degenerated vessel. (Fig. 56). There is a general proliferation of the surface endothelial cells which at the same time show degenerative changes. The majority of the superficial capillaries appear healthy. The walls of some of them are, however, thickened (Micro. spec. 57#), and proliferation of the endothelial cells of the perivascular canals can be seen at places. There is a little altered blood-pigment lying outside of some of them.

CASE No. 7

J. L., male, aged 38.

Case of phthisical insanity. Death from chronic phthsis.

Post mortem 14.3.94.

Naked-eye appearances. The dura appeared quite normal.

Microscopic appearances. Superficial horizontal sections show some localised proliferations of the endothelial cells of the surface, and also of those of the perivascular canals. (Micro. spec. 59). Many of the surface endothelial cells appear swollen and granular and are indistinctly stained. Some are irregular in outline. There are some deeply stained, slightly granular patches in the fibrous tissue near the surface. Transverse sections show an apparently normal dura. (Micro. spec. 60).

CASE No. 8

C. C., male, aged 50.

Case of advanced general paralysis. Patient sank gradually. He had no congestive attack for some months before death.

Post mortem 13th December 1894.

Naked-eye appearances. There was slight morbid adhesion of dura to calvarium. Slight general thickening, and opacity on holding up to light, about middle meningeal arteries. There was no membrane or rusty staining, but at places a faint yellow tint could be detected.

Microscopic appearances. In superficial horizontal sections there are to be observed especially indications of proliferative changes in the endothelial cells. Those of the inner surface show a slight general increase in their number, and also localised aggregations. Those of the perivascular canals also show a distinct increase which is especially marked at some places (micro. spec. 61). There are some small patches of degenerative change in the fibrous tissue of the dura characterised by deep staining and a slightly granular appearance. Many of these areas show proliferation of the superjacent endothelial cells. (Micro. spec. 62). Some

specimens show at places thin granular films on the surface. In micro. spec. 63+ one of these is seen which is continous with the wall of a perivascular canal. This specimen, which is an osmic acid preparation, also shows at the spot marked ++ distinct fatty changes either in the vessels or in their perivascular canals. Some minute haemorrhages are to be observed in this case (micro. spec. 62++). Concentric bodies occur; (micro. spec. 63++). The yellow tint referred to is probably due to a slight deposit of altered blood-pigment which may be seen in some preparations (micro. spec. 63+). Many of the vessels are much congested. There are probably no new capillaries.

Median horizontal sections show no morbid change.

CASE No. 9.

T. L., male, aged 46.

Case of advanced general paralysis. Patient died in a congestive attack. He had no previous similar attack during 18 months residence in asylum.

Post mortem on 15th December 1894. Typical signs of advanced general paralysis found in brain. Early pneumonia.

Naked-eye appearances. Slight morbid adhesion of dura to calvarium. Some opacity in neighbourhood of middle meningeal arteries on holding membrane up to light. No false membrane or rusty or yellow staining. Superficial vessels unduly prominent. Over body of the sphenoid and lesser wings there were numerous fine granulations. At the posterior edges of the lesser wings there was in addition a net-work of minute injected vessels. Some minute granulations could be seen at places over the whole of the inner surface.

Microscopic appearances. In superficial horizontal sections from vertex and from over sphenoid there are marked proliferative and degenerative changes in the endothelium of the surface and of the peri-vascular canals. The surface cells show a general increase in their number. (Micro. spec. 64+ Fig. 25) Those of the perivascular canals show more localised aggregations. micro. spec. 65+ Fig. 14). The

cell-plate of many of the former can be distinctly seen. It is mostly very granular and can often be observed to be in a state of disintegration. (Fig. 31). These degenerating cell-plates sometimes appear to form a thin granular sheet on the surface of the dura. (Micro. spec. 66+). In the nuclei of these cells the chromatin particles tend to be diffusely scattered, or localised in groups near the periphery instead of being arranged in an oval form in the centre. The aggregation of endothelial cells in the perivascular canals is at places very marked. There are some vessels surrounded by a hyaline material which may sometimes be observed to be continuous with a granular film on the surface, (micro. spec. 65 ##). Many of the superficial vessels are injected and some are varicose. In the sphenoidal dura these vessels are very numerous and much congested. Here there is to be observed a delicate fibrillated tissue immediately below the endothelium. There is no distinct evidence of the formation of new vessels. Osmic acid preparations show slight fatty changes in some of the vessels or perivascular canal-walls. Recent capillary haemorrhages are numerous. Concentric bodies occur in large numbers. Many of the granulations over the sphenoid as well as in other parts are distinctly due to groups of these bodies. Some interesting forms of developing

concentric bodies may be seen in some specimens. (Micro. spec. 67+ and 65+ Fig. 45). Some preparations show a small amount of altered blood-pigment lying near the surface.

Deep horizontal sections from the vicinity of one of the middle meningeal arteries show marked proliferative changes in the endothelial cells of the perivascular canals. (Micro. spec. 68+). These cells are undergoing degenerative changes resulting in the formation of small hyaline(?) bodies evidently essentially the same as concentric bodies. (Micro. spec. 68+ Fig. 51).

Case No. 10

G. B., male, aged 60.

Case of alcoholic insanity with delusional melancholia. Ill for about a year. Patient refused food and had to be fed with tube for about two months towards termination of case. Death occurred from drowning.

Post mortem on 24th December 1894.

Naked-eye appearances. The dura was moderately firmly adherent to the calvarium. Otherwise normal.

Microscopic appearances. The surface as seen in superficial horizontal sections appears for the most part perfectly normal. There are, however, some capillary haemorrhages. (Micro. spec. 69+) Some of the preparations also show distinct localised proliferation of the endothelial cells of the perivascular canals. (Micro. spec. 70+) There is also some altered blood-pigment in some of the perivascular canals (Micro. spec. 70++) No typical concentric bodies observed. There are, however, on the surface of some of the preparations rounded bodies which stain of a deep purple colour in haematoxylin and eosine preparations. They occur singly, in twos. or in groups. The form and colour

of the last suggest the simile of a mulberry. They sometimes have a capsule of a mucoid character stained of a lighter purple tint. (Micro. spec 71 A Fig. 52)

Case No. 11

H., female, aged 70.

Case of senile mania. Patient was operated upon in Edinburgh Royal Infirmary for epithelioma of rectum, shortly after which she became maniacal. She was removed to the Asylum and died there from exhaustion 16 days later.

Post mortem on 21st December 1894. Fatty degeneration of heart muscle with antemortem clotting in right chambers was found. There was marked brain atrophy and excess of fluid within the cranium. Naked-eye appearances. Slight morbid adhesion of dura to calvarium. Some general thickening and opacity on holding up to light around middle meningeal arteries. There were three small recent clots of dark blood about half an inch in diameter on the inner surface at the vertex on the left side. In the same region there was a soft gelatinous nodule of the shape and size of a small split pea. Over the body and lesser wings of the sphenoid the surface was somewhat rough and gelatinous-looking, and it was found that a delicate membrane could be scraped off in this situation. Elsewhere the surface was to a slighter degree granular and gelatinous-looking

but no membrane could be raised. After hardening in bichromate, however, it was found that a very delicate colourless coherent layer could be scraped off the inner surface of every part of the dura. Microscopic appearances. The membrane shows a very delicate fibrillar structure (evidently consisting of fibrin threads) which entangles very numerous cellular elements. (Micro. spec. 72) These consist of round cells, spindle cells, endothelial cells and red blood corpuscles. (Fig. 32 and 30) The majority of the cells are of the endothelial type. Red blood corpuscles are only numerous in places. There is only a very occasional capillary to be observed Concentric bodies are very numerous. (Micro. spec 73 , stained by Van Giesson's method) In some preparations there is a considerable amount of altered blood-pigment, mostly contained within leucocytes. Micro. spec. 74 is a preparation of one of the clots. It shows a localised recent blood effusion added to a membrane of the above characters. There is much altered blood-pigment around it. The gelatinous nodule shows the structure of an endothelioma. (Micro. spec. 75 & 76)

Superficial horizontal sections of the dura after removal of the membrane show very well marked proliferative changes in the endothelial cells both of the surface and of the perivascular canals.

(Micro. spec. 77+) Many of them are undergoing degenerative changes. The cell-plates of some of these cells are visible. (Fig. 31) Concentric bodies are very abundant and many developmental forms of them can be seen. (Micro. spec. 77+) There are also on the surface some "mulberry bodies" similar to those seen in previous case (Fig. 53) Micro. spec. 77 at spot marked x gives a good view of leucocytes lying in a perivascular canal. Some of the superficial vessels are considerably injected and small haemorrhages and spots of altered blood-pigment can be occasionally seen. Thereis no very distinct evidence of the formation of new capillaries. Micro. spec. 78 is a superficial horizontal section of the dura below the clot shown in Micro. spec. 74 . The point of the haemorrhage cannot be detected.

Case No. 12.

B. C., male, aged 52.

Case of senile insanity.

Post mortem 4th March 1894. Pulmonary phthisis.

Numerous cortical softenings in brain.

Naked-eye appearances. Dura appeared quite normal.

Microscopic appearances. The endothelial cells of the surface are probably slightly increased in number. Their intranuclear structure is quite obscured. There are no typical concentric bodies but small oval rounded bodies, mostly of a deep violet colour in haematoxylin and eosine preparations, somewhat resembling starch grains in form and frequently showing concentric markings, are numerous. (Fig, 50) Their development from the nuclei of endothelial cells can be distinctly traced. Some of them show a deep crimson centre. Opaque granular sheets can be seen on the surface at places. They are often continuous with strings of the same material which tends to contract and curl up. (Micro. spec. 79+) Some of the vessels are distinctly thickened. (Micro spec. 79++ x 80+) There are some grains of altered blood-pigment lying near the surface (Micro. spec. 79+) "mulberry bodies," similar to those described in the two previous cases are present in considerable numbers. Transverse sections reveal no abnormality. (Micro. spec. 81).

Case No. 13.

W., female, aged 62.

Case of senile insanity.

Post mortem 21st March 1894. Fatty heart with ante-mortem clotting in chambers.

Naked-eye appearances. The dura seemed perfectly normal.

Microscopic appearances. There are some slight proliferative changes in the endothelial cells of the surface and of the perivascular canals. They are specially well marked at some spots in the latter situation. Degenerative changes in the cells are not very marked, but some preparations show large numbers of small concentric bodies of the starch grain form. (Micro. spec. 82+) In this specimen they are lying on the outer or cut surface of the section, which position they have probably attained through being displaced from their original site in the course of preparation of the specimen. They were seen to be floating about while it was clearing in clove oil. There are no large concentric bodies of the typical form. Some of the vessels show distinct thickening of their walls. (Micro. spec. 83+) In some preparations there is much

altered blood-pigment lying outside of the vessels. Micro spec. 84) There are well marked degenerative patches in the fibrous tissue near the inner surface, having a granular appearance and staining deeply. (Micro. spec. 83+)

Transverse sections do not show these changes. In one, however, there can be seen a thickened vessel deep in the dura with a small haemorrhage round it. (Micro. spec. 85+).

Case No. 14

J. K., male, aged 39.

Case of advanced general paralysis. Patient was ill for three years. He had latterly repeated congestive attacks. He died from exhaustion and not in one of these seizures.

Post mortem 29th March 1894. Typical signs of general paralysis found in brain.

Naked-eye appearances. There was a very delicate false membrane with very slight rusty staining in the basal fossae of the left side. It was readily separable from the dura.

Microscopic appearances. In hardened and stripped preparations the membrane is seen to consist of a finely granular and slightly fibrillated material which is moderately cellular and permeated by very numerous, thin-walled, large and often varicose capillaries. The cells are probably mostly leucocytes or connective tissue corpuscles, but there are also many endothelial cells. The basis substance forms flattened net-works, several layers of which may be observed at places to be inaccurately superimposed. (Micro. spec. 86) Portions of these

net-works stain very deeply with haematoxylin. Many of the threads are joined to, and embrace, the vessels. Others have broken across and are contracting and curling up. There is a considerable amount of altered blood-pigment in the false membrane. There are some aggregations of cells which are probably endothelial in character. There is much fine granular matter which stains with eosine. There are some concentric bodies present. Micro. spec. 87 shows that they are unaffected by treatment with a strong solution of hydrochloric acid. Transverse sections of the dura with false membrane attached show a somewhat similar structure, but the features are less distinct. The line between the false membrane and the dura is not very sharp. The latter seems healthy. (Micro. spec. 88.)

Superficial horizontal sections of the dura from part where there was no false membrane show the surface in many places to be covered by a delicate film similar to those which form the membrane. (Micro. spec. 89) There is a distinct general proliferation of the surface endothelium and several localised dense aggregations of cells which seem to be of the endothelial type. The endothelium of the perivascular canals is also greatly proliferated and there is much blood-pigment lying outside

of some of the vessels. (Micro. spec. 90)
There are some degenerative patches in the fibrous tissue near the surface which are somewhat granular and stained deeply with haematoxylin.

CASE No. 15.

D.S., male, aged 25.

Case of phthisical insanity. Patient sank gradually. Post mortem on 30th March 1894.

Naked-eye appearances. There was a delicate faintly rusty stained false membrane on the inner surface of the dura overlying the upper surface of the brain, and equally developed on the two sides.

Microscopic appearances. Stripped preparations of the false membrane show at most places the structure of white blood-clot. Micro. spec. 91 Fig. 10). The fibrin threads are very distinct. The cellular elements, which are only moderately numerous are chiefly leucocytes, but there are in addition some endothelial cells. There are some dense aggregations of cells which are probably also of this character. Concentric bodies are numerous. (Fig 47). Some preparations show many newly formed large capillaries. (Micro. spec. 92).

Transverse sections of the dura show in most preparations a very delicate granular non-vascular false membrane on the inner surface. (Micro. spec. 93 Fig. 11). In others the false membrane is composed of red blood-clot. (Micro. spec. 94). The dural vessels are congested. There is no distinct cellular infiltration of the fibrous tissues.

Superficial horizontal sections of pieces of the dura from which the false membrane has been peeled off show that the superficial endothelial cells, and also those of the perivascular canals, are very markedly proliferated and degenerating. (Micro. spec. 95). Concentric bodies, especially of the starch grain form, are numerous. In these preparations the immediately subendothelial dural tissues show probably slight infiltration with leucocytes. Many of the superficial vessels are injected. There are some "mulberry bodies" present. They are stained of a brighter pink colour than usual.

CASE No. 16

M. H., female, aged 68.

Case of senile insanity. Death from Catarrhal pneumonia.

Post mortem on 18th March 1895.

Naked-eye appearances. There were slight granulations on the inner surface of the dura over the body and lesser wings of the sphenoid. Otherwise the dura seemed normal.

Microscopic appearances. The surface in superficial horizontal sections shows no very marked abnormality. (Micro. spec. 96). The endothelial cells stain faintly and are difficult to recognise. There is a small amount of altered blood-pigment occasionally to be seen in the perivascular canals. There are some concentric bodies, and groups of degenerated cells which stain of a deep purple colour occur occasionally on the surface. Distinct mulberry bodies are seen in some of the specimens.

In silvered preparations (Micro. spec. 97 and 98), the outlines of the endothelial cells are well shown. Some of the cell-plate tends to stain with silver as well as the ground substance. Concentric bodies appear of a more or less dark brown colour, (micro. spec. 97 Fig. 48). In micro. spec. 98 a granulation, probably formed of endothelial cells, is seen at the spot marked +. It contains some small concentric bodies.

CASE No. 17.

J. M., female, aged 68.

Case of senile insanity. Patient died from pneumonia following influenza.

Post mortem on 18th March 1895.

Naked-eye appearances. There were very numerous and large granulations over the sphenoid, and towards the posterior edge of the lesser wings there was a net-work of minute injected vessels. Otherwise the dura appeared normal.

Microscopic appearances. In superficial horizontal sections from the vertex the surface endothelium is very indistinctly brought out with haematoxylin and eosine staining. There are numerous concentric bodies. Some are of the starch grain form. There are also numerous mulberry bodies. Some of the vessels are considerably injected, and there are some small haemorrhages. (Micro. spec. 99).

In silvered preparations from the same region the outlines of the surface endothelial cells are not well brought out, but those of the cells forming the perivascular canal walls are very distinct. (Micro. spec. 100). In these preparations many distinctly thickened capillaries can be seen. (Micro. spec. 101). Some of them are lying on the cerebral side of the surface endothelium, though they can usually be traced to continuity with a vessel below

it. (Micro. spec. 102). Micro. spec. 103 shows an endothelial granulation around the cells composing which there is blackening with silver.

In superficial horizontal sections of the sphenoidal dura concentric bodies are present in very large numbers. *The granulations visible with the unaided eye are evidently entirely due to groups of these bodies. There are very numerous large dilated capillaries, most abundant in preparations from the posterior edge of the lesser wings. Numerous transition forms of concentric bodies are to be observed. (Micro. spec. 104). There are many hyaline, but slightly striated, branching rods which stain deeply with eosine. (Micro. spec. 105 ++). In their centre, or at their side, there is often a mass of material of the same appearance as the central portion of a typical concentric body. Surrounding some of the capillaries there is a similar hyaline but slightly striated sheath. It appears to be immediately outside of the capillary and internal to the endothelium of the perivascular canal wall(?)(micro. spec. 105+ Fig. 17). Similar but more purple and granular sheaths surround some capillaries which appear partly or completely occluded. There are also many concentric bodies of the starch grain form to be seen in this portion of the dura.

CASE No. 18

J. G., male, age unknown,- probably about 40.
Case of choreic insanity. Patient was admitted in a state of severe acute chorea. There was no history obtainable. Choreic movements continued until about six hours before death, when patient became comatose.
Post mortem on 5th November 1893.
Naked-eye appearances. There was a large recent blood effusion on the outer surface of the dura below the right parietal bone. Over the whole of the inner surface of the dura and outer aspect of the arachnoid (cerebral and spinal) there was a delicate but distinct, slightly rusty-looking false membrane which could be easily peeled off. There were large recent haemorrhages into the right tempero-sphenoidal lobe and right lobe of the cerebellum, the former bursting into the subdural space. There was also a small haemorrhage into the pons.
Microscopic appearances. The false membrane, examined in horizontal view, is seen to be for the most part non-vascular. It is composed of fibrin (the threads of which are somewhat indistinct), endothelial cells and leucocytes. (Micro. spec. 106). There are very numerous concentric bodies. Strings of the same material as that of which they are composed are also numerous, and there are some transition

forms to be seen. Disintegrating red blood corpuscles can occasionally be observed and also some grains of altered blood-pigment. In some parts of the false membrane there are very large dilated varicose capillaries. (Micro. spec. 107).

In transverse sections of the cerebral dura the only noticeable feature in addition to the delicate false membrane on the surface is marked injection of the dural vessels. Transverse sections from the cervical region show an inflection of the dura in which there are seen some concentric bodies (proving their development in situ). See micro. spec. 108+

Superficial horizontal sections of the cerebral dura after removal of the false membrane show in many places a perfectly normal structure. Other portions show marked proliferation of the endothelial cells both of the surface and of the perivascular canals. Some concentric bodies are present. There are numerous small patches in the fibrous tissue of the dura near the surface which are slightly granular and stain very deeply with eosine. (Micro. spec. 109+) In some specimens thin sheets and net-works of a granular and slightly fibrillated substance can be seen on the surface.

The stripped membrane from the spinal dura shows a similar structure to that from the cerebral dura. It is non-vascular. Concentric bodies are

extremely numerous, as well as strings of the same material as that of which they are composed. (Micro. spec. 110 Fig. 43, 37).

Superficial horizontal sections of the spinal dura with thin false membrane in situ show on the surface very numerous cellular elements. These are mostly endothelial cells, but are also largely leucocytes. There are very few vessels to be seen. Concentric bodies occur in very large numbers, and long strings of a similar material with various transitional forms may be observed. (Fig. 46 Micro. spec. 111). Some of the concentric bodies give evidence of their central and more deeply staining part being composed of several more or less rounded separate masses (Fig. 44). Some still more dense aggregations of cells occur, and in them concentric bodies are specially abundant. (Micro. spec. 112). There are some granules of altered blood-pigment. The intercellular substance is at places markedly fibrillated, suggesting the presence of coarse fibrin threads. Comparison with healthy duras, however, proves that this appearance is merely due to the normal elastic fibres of the spinal dura. There are in the false membrane numerous large irregular areas over which the intercellular substance stains more deeply with eosine than elsewhere. (Micro. spec. 113). In

specimens stained by Van Giesson's method these areas are brought out with remarkable distinctness, (micro. spec. 114), taking the fuchsine stain very deeply. In some preparations by this method the vessels are seen to be surrounded by a hyaline but slightly striated material. This in micro. spec. 115 at the spot marked + appears to be developing into concentric bodies.

(Micro. spec. 116+), others contain more or less disintegrated ones, while still others are filled with altered blood-pigment. There are some concentric bodies. In some parts the membrane consists of a single, or of several flattened layers of a fenestrated fibrinous-looking structure. (Micro. spec. 117+

Transverse sections show a very narrow moderately cellular false membrane on the inner surface. (Micro. spec. 118). There is no infiltration of the subjacent dura with round cells, and no injection of its vessels.

In superficial horizontal sections of the dura prepared after removal of the false membrane, and also of the portion which showed rusty staining without any distinct false membrane, there is over the greater part of the surface evidence of the formation of a delicate layer of new tissue (micro. spec. 119). In many parts of this little more than cellular elements can be made out. Others show a distinct layer of a granular and slightly fibrillated basis substance, which in some places is fenestrated, and occasionally also laminated. The cellular elements are made up of round and endothelial cells. There are numerous dense cellular aggregations consisting mainly of the latter. (Micro. spec. 120). These endothelial cells show numerous degenerative changes.

Many of them are swollen and granular, others are pale blue (in haematoxylin and eosine preparations), inflated-looking and sometimes distinctly vacuolated. Many transition forms leading up from the normal cell to concentric bodies of the starch grain form can be seen. Typical large concentric bodies also occur. The new tissue on the surface of the dura contains numerous vessels. They are not much dilated. Some of them have slightly thickened and granular walls. Others appear normal. There is a considerable amount of altered blood-pigment lying outside of them. Extravasated red corpuscles are numerous. There are very marked morbid changes in the original vessels of the dura near the surface. There are aggregations both of endothelial cells and round cells in their perivascular canals, which are further obstructed by collections of altered blood-pigment. (Micro.spec. 121). Some of the capillary walls are slightly thickened and granular. Fibrin threads can be seen within some of the vessels. The fibrous tissue of the dura shows near its surface numerous deeply stained slightly granular patches. Some hyaline-looking rods can be seen, the exact nature of which is doubtful.

CASE No. 20.

H., female, aged 24.

The case was clinically regarded as one of general paralysis. Patient had phthisis and at the post mortem it was found that death had been mainly due to this. There were several small tumour-like nodules and cysts in the brain, but none of the usual appearances found in general paralysis. Microscopic examination of the brain confirmed the view that the case was not one of this disease.

Naked-eye appearances. There was slight abnormal adhesion of the dura to the calvarium. There was adhesion between the dura and the arachnoid by recent semi-purulent lymph over an area about half an inch in diameter about the centre of the left parietal lobe. Otherwise the dura appeared normal.

Microscopic appearances. Superficial horizontal sections of the dura at some distance from the adhesion described above show at places on the surface a very delicate slightly granular and fibrillated film, apparently of a fibrinous character. (Micro. spec. 122+). In one specimen this film is specially distinct and its character beyond doubt. (Micro. spec. 123+). There is probably a slight increase in the number of the superficial endothelial cells. The vessels show no very distinct morbid change.

There are numerous small, granular, deeply staining patches in the fibrous tissue near the surface. (Micro. spec. 124†). There are some concentric bodies on or near the surface. (Micro. spec. 124††). There are some appearances which suggest that these degenerative patches are developing into concentric bodies. See especially microscopic specimen 125†

Transverse sections show a broad outer longitudinal layer. On the inner side of this there is a very narrow layer of tissue which suggests the presence of a false membrane. (Micro. spec. 126). Examination of several specimens, however, proves that it is a narrow inner layer of the dura. Some specimens show three distinct layers. The fibrinous membrane has evidently not remained attached. The vessels of the dura are not congested.

Superficial horizontal sections of the spinal dura show marked proliferation of the surface endothelial cells, and also of those of some of the perivascular canals. Concentric bodies are very numerous. There are many interesting imperfect forms. (Micro. spec. 127). There is probably no fribrinous film here, although the elastic fibres of the dura produce an appearance in places closely resembling one.

CASE No. 21.

J. G., male, aged 65.

Case of chronic melancholia. Patient was ill for several years. Death occurred from pneumonia. Post mortem on 14th May 1894.

Naked-eye appearances. The dura appeared normal.

Microscopic appearances. There is probably a slight increase in the number of the endothelial cells on the surface. Many of them show degenerative changes, and concentric bodies of the starch grain form occur in small numbers. There can be seen on the surface in many specimens a very delicate finely fibrillated and somewhat granular film, which in some places forms deeply stained net-works. (Micro. spec. 128+ and 130+). The superficial vessels are not specially congested. Many of them show fairly dense aggregations of cells around them in their perivascular canals. (Micro. spec. 129+). These cells are apparently both leucocytes and endothelial cells. There are some small recent haemorrhages into the perivascular canals (Micro. spec. 130++), but no altered blood-pigment is to be seen. Fibrin threads can be observed in some of the vessels (Micro. spec. 129++). There are some hyaline rods near the surface. Most of them appear to be continuous with strands of normal fibrous tissue. There are some deeply stained granular patches in the fibrous tissue near the surface. Transverse sections show no abnormality.

CASE No. 22.

J. G., female, aged 44.

Case of long standing dementia. Death occurred from phthisis.

Post mortem on 5th February 1894.

Naked-eye appearances. There was an extremely delicate colourless false membrane over the greater part of the inner surface of the dura overlying the superior and lateral aspects of the hemispheres. It could be easily scraped off, but could not be raised as a coherent layer.

Microscopic appearances. Even after hardening it was almost impossible to strip a coherent membrane from the surface. Only one small specimen was obtained. (Micro. spec. 131). It shows a granular and fibrillated structure, moderately cellular, with a small number of dilated vessels. The cells are mostly endothelial in character but leucocytes are also present.

Superficial horizontal sections with false membrane in position show in many of the specimens very delicate granular and fibrillated sheets on the surface. (Micro. spec. 132). In some places these are in the form of a net-work, many of the threads of which have a hyaline appearance. (Micro. spec. 133). The endothelial cells of the surface are somewhat increased in number, and they show degenerative

changes. Some of them are clearly developing into concentric bodies of the starch grain form. This is well seen in the last specimen. The vessels are not specially congested. Many of them show slight aggregations of cells upon their walls,- both endothelial cells and leucocytes. The walls of many of the vessels are markedly thickened and granular, staining deeply with eosine. (Micro. spec. 132+). No altered blood-pigment can be seen. There are some marked patches of granular change in the fibrous tissue near the surface.

In transverse sections a delicate false membrane is seen at places on the inner surface. There is no cellular infiltration of the tissues of the dura and no congestion of its vessels. (Micro. spec. 134).

CASE No. 23.

H. L., male, aged 38.

Case of early general paralysis. Patient had a congestive attack two months before death. He remained in a somewhat stuporose condition subsequently. He was found one evening in an unconscious state and died six hours afterwards.

Post mortem on 18th August 1894. Death was found to be due to a large haemorrhage into the subdural space.

Naked-eye appearances. Extending throughout the whole of the left side of the subdural space including the cerebellar fossa, there was a large blood-clot. It was fully half an inch in thickness at places. On each side it was covered by a thin, condensed, almost colourless membrane which separated it from the dura on the one side and the arachnoid on the other. On the under surface of the left tempero-sphenoidal lobe there was a minute opening,- of a size just sufficient to admit a common pin,- in a small vessel from which the blood effused into the subdural space had evidently escaped. For half an inch around this point there was some blood effusion into the arachnoid spaces. The vessel did not appear to be otherwise diseased. The dura on the right side seemed healthy.

Microscopic appearances. The false membrane is in

no part thin enough to allow of examination in horizontal view as a stripped specimen. Horizontal or oblique sections of the thickest part, which appeared to the naked-eye as a red blood-clot, show numerous red blood corpuscles many of which are disintegrating, granular debris resulting from this disintegration, leucocytes, endothelial cells, fibrin threads and some penetrating capillaries. (See micro. spec. 135, and also no. 136 which is a superficial horizontal section of the clot.) Horizontal sections of the firm colourless membrane underlying a thinner portion of the clot show a somewhat dense structure of fibrin threads containing very numerous cellular elements. These are leucocytes, endothelial cells, and red blood corpuscles. (Micro. spec. 137). The endothelial cells are most numerous. They show degenerative changes,- chiefly swelling up into pale bodies from which the deeply staining intranuclear network and nucleolus have disappeared, vacuolation and the formation of concentric bodies of the starch grain form. The protoplasm of some of these endothelial cells may be distinguished. It is mostly somewhat granular. The leucocytes at places form dense aggregations. Red blood corpuscles are only evident in some parts. It can be seen that they are being absorbed by leucocytes, many of which also are filled with altered blood-pigment. (Micro. spec. 138.)

Some typical large concentric bodies are present.

Superficial horizontal sections of the dura after the blood-clot had been peeled off, show shreds of the latter still adhering to the surface and presenting the same structure as that described above. (Sp. 139) There is a general increase in the number of the endothelial cells of the surface and of the perivascular canals. Some apparently new vessels can be seen near the surface penetrating the clot.

Transverse sections of the dura after removal of the red blood-clot show on the inner surface a narrow, dense, longitudinally striated, moderately cellular band without vessels,- representing the dense coagulum already described. (Sp 140) Otherwise there is no abnormality.

CASE No. 24.

A. M., female, aged 12.

Case of developmental general paralysis. This patient was for a short time an inmate of the Royal Edinburgh Asylum, but was taken home before death. The post mortem was performed by Dr W.R.Dawson, to whom I am indebted for the material for microscopic examination. Along with Dr John Thomson he has reported the case in "The Lancet" of 16th February last. Dr Dawson's report on the naked-eye appearances of the dura is as follows. "The skull-cap, the inner and outer tables of which were thickened (partly at the expense of the diploë), had contracted adhesions with the dura mater, especially along the superior longitudinal sinus. The sinuses contained red and white blood-clot and were engorged, as were the veins opening into them from the pia mater, whilst the dura mater itself was everywhere thickened, and in some places was separated from the bone by a false membrane, the latter a somewhat rare condition, constituting the pachymeningitis externa of German writers. In addition to this, however, the dura mater over the whole extent of the cranium was lined with a false membrane, which varied greatly in thickness, being merely a thin film in the middle cranial fossa, whereas over the parietal regions it was fully a quarter of an inch thick in

places. This membrane was adherent to the arachnoid all over the vault, though not very firmly, except in the part corresponding to Broca's lobe, where a fibrous band was noted. It left the cerebellum uncovered, but was observed to pass downwards beneath the pons and medulla so as to invest the spinal cord also." I would add that the subdural false membrane showed very marked lamination in its thicker portions, permitting when hardened of its being split into layers.

Microscopic appearances. Transverse sections of the dura and false membrane taken from various parts present in each case a similar structure (Sp.141) The false membrane is composed of granulation tissue or more perfect fibrous tissue with somewhat scanty cellular elements. There are large areas in which no vessels can be seen. The fibres run for the most part parallel to the dural surface and have a wavy outline. In addition to connective tissue corpuscles and leucocytes there are numerous cells of the endothelial type. Some collections of granules of haematoidin are present, but the false membrane is for the most part free from this deposit. Recent blood-extravasations can be seen in many places. They are sometimes of a diffuse character. (Micro. spec. 142). In some preparations towards the free edge of the false membrane the fibrous

elements have undergone a peculiar degenerative change, swelling up into a glassy substance.(Micro. Spec. 143). Micro. spec. 144 shows a false membrane developed on both sides of the dura. It has the same characters in the two situations. Micro. spec. No. 145 shows a recent blood-clot in the centre of the dura. In specimen No. 146 in the same position there is seen some new tissue exactly like that of which the false membrane is composed. In specimen No. 147 at the spot marked + it can be observed that the lines along which the false membrane could be split into layers are composed of a narrow band of looser and more cellular tissue than that which forms its general structure. Numerous capillaries are frequently to be observed in these bands. At the spot marked ++ in the same preparation there is seen a recent haemorrhage from this tissue. The dural tissues as seen in these transverse sections appear healthy, except that the vessels are greatly dilated and injected with blood.

Superficial horizontal sections of portions of the dura from which the false membrane has been peeled off show some shreds of the latter remaining on the surface. There is a general increase in the number of the endothelial cells both of the surface and in the perivascular canals. Many of the

original superficial dural vessels show somewhat thickened walls. (Micro. spec. 148). In some preparations there is much altered blood pigment near the surface. There are some concentric bodies both of the typical kind and of the starch grain form.

CASE No. 25.

M. O., female, aged 70.

Case of senile insanity. Death occurred from inflammatory gangrene of left foot and leg. There was no rise of temperature.

Post mortem on 30th January 1895.

Naked-eye appearances. The dura was very firmly adherent to the calvarium. Over the entire inner surface there was a peculiar opaque silvery appearance. It was more noticeable at some places than at others, being most marked at the vault. There was no other abnormality.

Microscopic appearances. In superficial horizontal sections the silvery appearance is seen to be due to masses of transparent colourless globules. They vary considerably in size, some being very small, others being about the diameter of a red blood-corpuscle, and some considerably larger. In some specimens they are scattered all over the surface, but are at the same time collected into ill-defined masses which are elongated in the direction of the connective tissue fibres. (Micro. spec. 149). Immediately subjacent to these masses the fibrous tissue is usually deeply stained. Careful examination enables one to trace these globules from the surface endothelial cells which, wherever visible, show marked morbid changes of this kind. The staining reaction to haematoxylin and eosine is in all the

tissue elements unusually diffuse so that many of the appearances are wanting in distinctness. It can be seen that a similar change is taking place in the endothelial cells of the perivascular canal walls. In specimen No. 150 at the spot marked + these cells can be seen to be swollen up into clear crystalline-looking bodies which fill the perivascular canal and compress the contained vessel. Some small recent blood-extravasations can be seen associated with these morbid changes. A still further change can be seen in many places to be taking place in these crystalline cells both on the surface and in the perivascular canals. It consists in their tending to coalesce, and in their becoming somewhat less transparent and assuming a strong affinity for eosine in haematoxylin and eosine preparations. Long masses of this material can be seen to fill some of the perivascular canals. (Micro. spec. 150, 151 and 152). It can further be seen that this material develops into concentric bodies which are numerous. They have, however, a somewhat unusual appearance, being much more transparent than they generally are and having a slightly yellow tinge. (See micro. spec. 150). In micro. spec. 151 at the spot marked + one of these concentric bodies is seen lying below a dural vessel. The walls of the superficial

vessels are for the most part distinctly thickened and tend to have an irregular outline. They stain unusually deeply with haematoxylin. In specimens fixed with osmic acid and afterwards hardened in potassium bichromate solution it is seen that the crystalline globules do not give any fatty reaction. (Micro. spec. 153). In micro. spec. 154, which is a similar preparation, the advanced changes in the perivascular canals already described are well seen. In some preparations there are seen on the surface delicate shreds of fibrinous and cellular membranes, in some parts showing red blood corpuscles. (Micro. spec. 155). In the osmic acid preparations it can be seen that there are in these false membranes some globular masses which give a fatty reaction.

CASE No. 26.

J. F., male, aged 23.

Case of dementia following adolescent insanity.

Death occurred from phthisis.

Post mortem on 23rd January 1895.

Naked-eye appearances. The inner surface of the dura had at places a slightly silvery appearance. Otherwise it seemed normal.

Microscopic appearances. In superficial horizontal sections stained with haematoxylin and eosine the endothelial cells are unusually indistinct, over considerable areas they appear to be entirely shed from the surface. Most of those that remain show marked degenerative changes. They appear vacuolated and are often shrivelled. The morbid condition seems to be essentially the same as that described in the previous case, but it is much less distinct. (Micro. spec. 156+). Many of the cells are converted into crystalline-looking bodies. In micro. spec No. 157+ a mass composed of such cells is seen on the surface. The endothelial cells of the perivascular canals have in many places undergone the same change, obliterating by pressure the contained capillary in the same way as was observed in the previous case. In these perivascular canals there is occasionally seen a thin-walled dilated capillary which is probably of new formation. (Micro. spec. 158+). Some

granular and deeply stained patches occur in the fibrous tissue, but they are not nearly so marked as in the previous case. No concentric bodies, altered blood-pigment, or fibrillated sheets are observable.

CASE No. 27

W. M., male, aged 73.

Case of senile insanity. Death occurred from gangrene of lung.

Post mortem on 20th February 1895.

Naked-eye appearances. The dura was markedly adherent to the calvarium. It showed some general thickening. There was some rusty staining without the presence of a separable membrane on the inner surface in the left anterior fossa, and in the other basal fossae there were some large dark clots of recently extravasated blood. There were very numerous minute spots of recent haemorrhage on the inner surface of the dura overlying the convexity of both hemispheres. There was no evidence of any haemorrhage from the vessels of the pia-arachnoid. In the bichromate hardened dura the inner surface had a general gelatinous appearance.

Microscopic appearances. In superficial horizontal sections of the dura from the vault the endothelial cells of the surface and of the perivascular canals show very marked morbid changes. They have evidently undergone marked proliferation followed by a degenerative change similar to that which is observed in the two previous cases, but it is here developed to an extreme degree. Only a small number of healthy-looking endothelial cells can be seen.

Others are markedly vacuolated, the stained portion having at the same time a shrivelled appearance. The majority of them have undergone a still further change, being represented by only a vitreous-looking globule. (Sp. 159 + & 160 ++) The same change is seen to be taking place in the endothelial cells of many of the perivascular canals. See micro. spec. No. 160 at the spot marked. + In places there are numerous new dilated capillaries. Recent haemorrhages are abundant. Numerous hyaline cords can be seen near the surface. (Micro. spec. 159 ++ and 160 +++). There are also numerous concentric bodies and some interesting developmental forms may be observed. (Micro. spec. 160 ++). In specimen No. 159 at the spot marked ++ , as well as in the immediate vicinity, some vessels may be seen with a hyaline band on each side of them. Specimen No. 161 , which is a superficial horizontal section (with the outer or cut surface uppermost) of a piece of the dura fixed in osmic acid and afterwards fully hardened in bichromate solution, shows the extent to which fatty changes occur. There are some large black globules on the inner surface, and many cells can be seen with minute black droplets in them. These appear to be mostly endothelial cells of the surface and of the perivascular canals.

CASE No. 28.

J. M., male, 49.

Case of long standing secondary dementia following adolescent mania. Death occurred from phthisis. Post mortem on 16th January 1895.

Naked-eye appearances. There were some very small areas of recent blood effusion on the inner surface of the dura near the superior longitudinal sinus,- two on the right side and one on the left. There was some general congestion of the inner surface. The congested vessels appeared as arborescent markings. They were specially prominent over the body and lesser wings of the sphenoid.

Microscopic appearances. Over large areas of the inner surface there is a delicate layer of new and highly vascular tissue. It is for the most part lowly organised granulation tissue, but in some places it is evidently merely a fibrinous coagulum which is becoming vascularised. It contains very numerous leucocytes, endothelial cells and red blood corpuscles and also a small amount of altered blood-pigment and granular débris. Many of the vessels are greatly dilated and varicose, and filled with red corpuscles. There are numerous recent haemorrhages from them. Others show marked thickening of their walls with more or less complete occlusion of

their lumen. The thickened wall often has a hyaline appearance. These degenerated new vessels are in places very numerous. (Micro. spec. 162).

The surface endothelial cells are greatly increased in number and they show degenerative changes. The original vessels of the dura present very marked morbid changes. Their walls are more or less thickened and granular, and some of them show varicosity. In many places the endothelial cells of the perivascular canals are markedly proliferated. Many of these vessels can be seen to be partially or completely occluded. Haematoidin granules lie outside of some of them. Micro. spec. No. 163 at the spot marked + shows a good example of a degenerated superficial dural vessel. Specimen No. 164 at the spot marked + shows a similarly degenerated vessel with a new and greatly dilated capillary lying alongside of it. Specimen No. 165 at the spot marked + shows another good example of a degenerated and occluded dural capillary, lying over which there is a network of new capillaries. These new capillaries often appear to be spreading over the surface where the original vessels are occluded, without the previous occurrence of haemorrhage. See micro. spec. No. 166 Some of the original dural vessels show on each side of them a hyaline band the nature of

which is doubtful. See micro. spec. No. 167 at the spot marked, as well as at other spots in the immediate neighbourhood. Concentric bodies occur in moderate numbers. Deeply staining granular patches occur in the fibrous tissue. Superficial horizontal sections of portions of the dura fixed overnight in osmic acid and afterwards hardened in bichromate solution, show absence of any very decided fatty changes. Some cells, however, can be seen with a few black granules in them. (Micro. spec. 168).

CASE No. 29.

Child, aged nine months.

Case of severe rachitis. Died in Royal Hospital for Sick Children, Edinburgh.

Post mortem on 29th May 1894.

Naked-eye appearances. On the inner surface of the dura over the convexity of the left hemisphere there was a recent blood-extravasation about an inch and a half in diameter. The clot was thin and dark red in the centre, but pale towards the edges. It stripped off quite readily as a coherent film. The surface of the dura below the clots was intensely injected.

Microscopic appearances. Only the edge of the clot was sufficiently thin for examination as a stripped preparation. This portion of it shows the following structure. (Micro. spec. 169). There is a dense network of fibrin threads entangling in some parts very numerous cellular elements. In others these are scanty. The cells are mostly leucocytes, but numerous endothelial cells may also be detected. Red blood corpuscles are in parts entirely absent. At others they are present in considerable numbers. They can be seen to be breaking down into granular débris,- partly of a deep brown colour, and partly colourless but staining deeply with eosine. Some of the endothelial cells can be observed in process

of degeneration into concentric bodies of the starch grain form. One or two delicate capillaries may be observed, betokening commencing vascularisation of the clot. In specimen No. 171 there is seen a long, deeply stained, slightly striated rod of a substance closely resembling coagulated fibrin. It can be seen to form a flat expansion at one end. A superficial horizontal section of the clot at its thickest portion,- presenting that is to say its inner or cerebral aspect,- shows on the surface numerous cells of the endothelial type. (Micro. spec. 172).

Transverse sections through the thickest portion of the false membrane show that in that part it is largely composed of red corpuscles (Micro. spec. 173). The dural vessels are somewhat congested. Specimen No. 174 shows one of these the contents of which appear to be continuous with the blood-clot on the surface. Some transverse sections show marked aggregation of cells in or around the vessels near the surface. (Micro. spec.No. 175).

Superficial horizontal sections of the dura beneath the clot, made after stripping the false membrane off, show numerous fibrinous, more or less cellular sheets and threads still adhering to the surface. At places they can be seen to form a network. (Micro. spec. 176). Apart from these fibrinous shreds the cellular elements appear numerous in

looking down upon the section, but with the high power it can be made out that they are mainly lying on the surface. They consist both of leucocytes and endothelial cells. Many of the latter show degenerative changes. Some of the superficial vessels show aggregations of leucocytes within them or in their perivascular canals. Probably many of the cells in the latter situation are, however, endothelial in character.

CASE No. 30

Child, aged 18 months.

Case of advanced rachitis. Died in Royal Hospital for Sick Children, Edinburgh.

Post mortem 31st May 1894.

Naked-eye appearances. There were two thin films of recent blood-clot on the inner surface of the dura overlying the convexity of the hemispheres,- one on each side. They were from one to two inches in diameter. The centre of each was of a dark red colour, the periphery much lighter. They could be stripped off readily from the dura, leaving a somewhat congested-looking surface. This congestion was confined to the position of the false membranes.

Microscopic appearances. The false membranes present in horizontal view the same characters as in the previous case.

In transverse sections it is seen that the clot at its centre is composed of two layers,- one next the dura which contains densely packed red blood corpuscles and leucocytes, the latter being most numerous near the dura,- and another, lying to the cerebral side of this, composed almost entirely of fibrin threads. The former layer gradually fades away at the sides, while the latter is continued to a much greater distance on the surface of the dura. (Micro. spec. 177).

Superficial horizontal sections of the dura below the clot show at places some shreds of the false membrane. There are numerous dense aggregations of leucocytes on the surface. (Micro. spec. 178). Numerous endothelial cells can also be seen, but it is doubtful if they are more abundant than normal. The superficial vessels are marked out by small dense aggregations of leucocytes. These are in many instances confined to the interior of the vessel, the perivascular canal, remaining empty. In other cases, however, they have passed into the perivascular canal in large numbers. (Micro. spec. 179.)

CASE No. 31

J. R., male, aged 19.

Case of sarcoma of thigh.

Post mortem at Royal Edinburgh Infirmary on 15th December 1894.

Naked-eye appearances. The dura was normal.

Microscopic appearances. In superficial horizontal sections there is seen some degree of cellular aggregation around many of the superficial vessels. The cells are probably chiefly leucocytes, though some of them are endothelial. (Micro. spec. 180) Some of the capillaries appear somewhat granular and thickened. A few may be seen which have a distinct hyaline sheath. (Micro. spec. 181). There are some small capillary haemorrhages (Micro. spec. 182), and some masses of fibrin are also to be observed outside of the vessels. (Micro. spec. 183). There are in some of the preparations numerous deeply stained granular patches in the fibrous tissue near the surface. (Micro. spec. 184). There are some structures which are evidently forms of concentric bodies. (Micro. spec. 185). An occasional "mulberry body" is to be observed on the surface. The endothelial cells appear for the most part quite healthy.

CASE No. 32

M. B., male, aged 56.

Case of middle ear disease; operation; death.

Post mortem at Royal Edinburgh Infirmary on 16th December 1894.

Naked-eye appearances. "The dura over the right petrous bone had just been perforated and some blood-clot lay below it." Otherwise healthy.

Microscopic appearances. There are in some of the preparations (superficial horizontal sections) numerous deeply stained and granular patches in the fibrous tissue. (Micro. spec. 186). Some of the vessels contain numerous leucocytes. Otherwise the dura seems normal.

CASE No. 33.

J. F., male, aged 53.

Case of fracture of the base of the skull.

Naked-eye appearances. The dura over the hemispheres was normal. At the base of the skull there was a fissured fracture with some haemorrhage into the subdural space in its vicinity. The hardened dura from the vertex seem somewhat congested but otherwise normal.

Microscopic appearances. A layer of coagulated fibrin can be seen on the surface at places. Sometimes it is mixed up with red corpuscles. (Micro. spec. 187). In this same specimen at the spot marked ++ there is a fibrillated sheet which may either be of the same nature or a subendothelial layer of delicate connective tissue. Around some of the vessels the endothelial cells appear to be somewhat excessive in number. (Micro. spec. 187). There are some small capillary haemorrhages. Some of the preparations show deeply stained and granular patches in the fibrous tissue.

CASE No. 34

J. G., male, aged 39.

Case of compound fracture of the skull.

Post mortem at Royal Edinburgh Infirmary on 21st December 1894.

Post mortem appearances. There was an extensive lacerated wound of the scalp, the tissues around which were swollen, oedematous and infiltrated with blood. There was an irregular aperture in the cranium about one inch in diameter, with a hernia cerebri protruding. There was laceration of the frontal lobe and diffuse haemorrhage into the arachnoid over the hemispheres.

Naked-eye appearances of hardened dura. It seemed slightly thickened. On the inner surface there were some shreds of a thin, opaque, somewhat friable but still coherent false membrane.

Microscopic appearances. The false membrane examined in horizontal view is found to consist of fibrin which entangles very numerous cellular elements, and much granular débris. Some of the cells have one, two or more deeply stained nuclei. Others are very pale rounded or oval cells. Some red blood corpuscles can be seen. Many of them are evidently breaking down into débris.

In superficial horizontal sections shreds of a

false membrane of the same kind can be seen lying on the surface at many places. (Micro. spec. 188). The pale cells already mentioned are very numerous. They have a large nucleus which stains very faintly with eosine in haematoxylin and eosine preparations, while the surrounding protoplasm stains rather more deeply with eosine. The endothelial cells around the vessels and on the surface, which everywhere show marked proliferative changes, have exactly the same appearance. Some sections of the dura devoid of false membrane show, however, little or no abnormality. In others there is slight infiltration of the tissues with leucocytes, while in others again all the cellular elements stain exceedingly faintly. In the latter especially, the surface and deeper parts are crowded with fine and coarse granular matter, a portion of which stains with eosine and a portion lightly with haematoxylin.

CASE No. 35.

W. C., male, aged 53.

Case of phthisis and pneumothorax.

Post mortem at Edinburgh Royal Infirmary on 17th December 1894.

Naked-eye appearances of hardened dura. There were several small light brown areas seen through the inner surface. Otherwise normal.

Microscopic appearances. The brown spots are seen to be due to somewhat recent haemorrhages infiltrating the tissues. One of these areas is seen in specimen No. 189†. It shows much granular matter, fibrin threads and breaking-down red corpuscles. There are delicate but distinct fibrinous sheets lying on the surface at other parts. They may be seen to be contracting into networks and hyaline strands. (Micro. spec. 189‡‡). In the same specimen at the spot marked ††† there is seen a hyaline rod which is apparently continuous at one end with the normal fibrous tissue of the dura. There are no very distinct endothelial changes. There are some granular patches in the fibrous tissue.

CASE No. 36.

M., female, aged 53.

Case of fracture of vault of skull.

Post mortem at Edinburgh Royal Infirmary on 14th January 1895. There was a large extra-dural haemorrhage from the left middle meningeal artery, and laceration of the tip of the left tempero-sphenoidal lobe. There was some fluid blood on the inner surface of the dura.

Naked-eye appearances of hardened dura. There appeared to be no abnormality.

Microscopic appearances. There are to be observed on the surface, examined in superficial horizontal sections, numerous shreds of a fibrinous false membrane containing abundant leucocytes, some endothelial cells, some red corpuscles and granular débris. Distinction can in this case be made fairly easily between such a fibrinous shred and the looser veil of fibrous tissue which sometimes lies below the endothelium. In specimen No. 190 at the spot marked +++ the normal surface is shown. In the same specimen at the spot marked + some shreds of a fibrinous false membrane are seen lying on the surface of a similar structure. Some of the vessels show aggregation of leucocytes within and around them. (Micro. spec. No. 190++). There are probably some proliferative changes in the surface endothelial cells at some places. There are some slight granular degenerative patches in the fibrous tissue.

CASE No. 37

J. F., male, aged 57.

Case of aortic incompetence, and aortic aneurism with rupture into oesophagus. The hardened dura appeared quite healthy.

Microscopic appearances. For the most part the surface appears quite normal, as seen in superficial horizontal sections. There is at places, however, a small amount of granular debris, and also some delicate fibrinous shreds. One specimen shows two small areas of a distinct false membrane composed of fibrin and granular debris, mixed up with very numerous endothelial cells and some red blood corpuscles. (Micro. spec. No. 191). The smaller of these two areas is distinctly associated with a recent blood extravasation. In specimen No. 192 at the spot marked + a number of the surface endothelial cells are seen surrounded by granular debris which appears to have arisen from disintegration of the cell-plates. At the spot marked ++ in the same specimen what is probably a normal subendothelial layer is well shown. There are some slight granular degenerative patches in the fibrous tissue.

CASE No. 38.

R. D., male, aged 26.

Case of "Leucocythaemia? Purpura haemorrhagica. Double Bell's paralysis."

Post mortem at Edinburgh Royal Infirmary on 5th March 1895. The dura mater showed numerous small distinct haemorrhages. These were far more plentiful on the inner than upon the outer surface. The pia and brain were normal.

Naked-eye appearances of hardened dura. The inner surface looked slightly granular and seemed to have a very delicate coating of a transparent substance upon it, which, however, could not be raised as a coherent membrane.

Microscopic appearances. The surface endothelium over extensive areas has undergone a peculiar degenerative change (micro. spec. No. 193†). The cells are greatly swollen and have a glassy appearance. All trace of the original structure has in most of them quite disappeared. In the early stage of the change the nucleus is filled with small globules of the same kind. This glassy substance does not stain with eosine in haematoxylin and eosine preparations, but its outline is demarcated by the haematoxylin stain. Over other areas the endothelial cells of the surface seem fairly healthy. The

vessels are very difficult to see in areas which are affected by the degenerative change but it can be made out that the endothelium of the perivascular canals has undergone a similar change, leading to compression of the contained vessel. (Micro. spec. No. 193#). There are on the surface numerous thin shreds of false membrane. It is composed of fibrin, leucocytes, endothelial cells (normal, or in various stages of degeneration), red blood corpuscles and granular débris. (Micro. spec. No. 194†). Some concentric bodies are present.

CASE No. 39

M., female, aged 50.

Case of fracture of femur, congestion of lungs, and fatty heart.

Post mortem at Royal Edinburgh Infirmary on 4th February 1895. There was oedema of the brain. The convolutions were atrophied and there was considerable increase of subarachnoid fluid. The kidneys showed diffuse cirrhosis.

Naked-eye appearances. The hardened dura showed no abnormality.

Microscopic appearances. In superficial horizontal sections stained with haematoxylin and eosine there are near the surface numerous cords (corresponding in diameter to the normal superficial capillaries), which are deeply stained with eosine, and most of which show a fine longitudinal striation. The distribution of many of them corresponds to that of the normal dural capillaries, which are usually scanty where these cords are abundant. They are seen at almost any part of specimens No. 195, 196, 197 They are mostly embedded in the fibrous tissue, but many are distinctly above the general surface level. The appearance of many of them suggests strongly that they are thickened capillaries with obliterated lumen. (See especially micro. spec. No. 195+)

Micro. spec. No. 196 shows at the spot marked + a vessel with similar but narrow cords lying on each side of it, or surrounding it. In other cases an appearance is presented which suggests that these cords are merely modified strands of fibrous tissue. There are numerous recent capillary haemorrhages and in some parts there are delicate fibrinous shreds and net-works scattered over the surface. (Micro. spec. No. 197 +). The cords of these net-works appear as a rule of a more purple hue than the striated cords already described, but they can often be traced into continuity with the latter. They expand at places into delicate fibrinous sheets on the surface. They can often be seen also to have been in process of contracting and curling up. There is a general increase in the number of the surface endothelial cells, and occasionally dense cellular aggregations, probably of the same nature can be seen. Concentric bodies are somewhat numerous, - both the typical large ones and those of the starch grain form. Many of the former seem to have a distinct developmental relationship to the striated cords, being embedded in them or lying alongside them.

CASE No. 40

R. Y., male, aged 41.

Case of chronic pulmonary phthisis.

Post mortem at Royal Edinburgh Infirmary on 4th February 1895. The hardened dura appeared normal to the naked-eye.

Microscopic appearances. There are numerous delicate shreds and cords of a fibrillated material on the surface. They contain numerous red blood corpuscles at places. (micro. spec. 198+). In specimen No. 199 at the spot marked + one of these striated cords is seen to be continuous with a similar material surrounding a superficial capillary,- that is to say lying in its perevascular canal. There are some concentric bodies present, both of the typical kind and of the starch grain form. There are some granular degenerative patches in the fibrous tissue. The surface endothelial cells show a distinct increase in their number at places. The vessels do not show any very distinct changes.

CASE No. 41

E. M., female, aged 35.

Case of mitral stenosis, and chronic venous congestion of organs. The hardened dura appeared normal to the unaided eye.

Microscopic appearances. Except for evidence of slight proliferative changes in the endothelium of some of the perivascular canals, and a few small granular patches in the fibrous tissue, this dura appears normal. (Micro. spec. No. 200).

CASE No. 41

E. M., female, aged 35.

Case of mitral stenosis, and chronic venous congestion of organs. The hardened dura appeared normal to the unaided eye.

Microscopic appearances. Except for evidence of slight proliferative changes in the endothelium of some of the perivascular canals, and a few small granular patches in the fibrous tissue, this dura appears normal. (Micro. spec. No. 200).

CASE No. 42

B. M., female, aged 59.

Case of malignant structure of colon, perforation and peritonitis.

Post mortem at Edinburgh Royal Infirmary on 6th February 1895.

Naked-eye appearances of hardened dura. The inner surface had a slightly gelatinous look, and appeared finely granular especially at spots.

Microscopic appearances. There is a very marked general increase in the number of the endothelial cells both of the surface and of the perivascular canals. There is a marked increase in the number of vessels. Scattered over the surface there are very numerous areas over which there are fibrinous films and shreds mixed up with endothelial cells, round cells, some red blood corpuscles and granular débris. At one spot there is a recent haemorrhage of considerable size. (Micro. spec. No. 201 +). In specimen No. 202 at the spot marked + there is seen a fibrinous film with a capillary passing into it. Hyaline, but distinctly striated cords can be seen radiating from it. There are large areas over which there are on the surface networks of vessels with thickened walls and partially occluded lumen, which have sheaths of the same hyaline but

longitudinally striated material. This can frequently be seen to form independent cords and to expand into thin striated veils or distinctly fibrinous films. (micro. spec.No.202" and No.203'). A large number of the vessels to be seen near the surface are new capillaries, judging from their number, distribution and absence of perivascular canals. There is no altered blood-pigment. Concentric bodies are somewhat numerous. The endothelial cells show some degenerative changes.

CASE No. 43.

J. T., female, aged 55.

Case of fracture of the base of the skull. The left hemisphere was covered with a thin layer of dark clotted blood lying in the subdural space. There was laceration of the left tempero-sphenoidal lobe. At the base there was well marked leptomeningitis of a suppurative type.

Naked-eye appearances of hardened dura. The piece of dura sent showed a very finely granular appearance on the inner surface. Otherwise it seemed normal.

Microscopic appearances. Over the greater part of the inner surface there are delicate fibrinous shreds and networks mixed up with very numerous endothelial cells. (Micro. spec. 204+). There is everywhere marked proliferation of the surface endothelial cells, while at places they form somewhat dense aggregations. Many vessels can be seen on or above the surface with thickened walls and completely or partially obliterated lumen, and enclosed in a hyaline but longitudinally striated sheath. (Micro. spec. No. 204++). There are some concentric bodies.

AUTHOR'S CONCLUSIONS

In the recent paper published by Dr Middlemass and myself (156) on "Morbid Conditions of the Dura Mater," in the introductory paragraph to the section on subdural membrane formation, we insisted upon the great complexity and difficulty of the problems which arise in the consideration of the subject, and expressed the opinion that in the case of many of them a solution could not yet be looked for. That the subject is of this nature should be evident enough when one considers the remarkable disproportion between the extent of the literature upon it and the amount of information which that literature gives concerning the morbid tissue changes which occur in the process of the disease; but if any doubt remains as to the fact it must be dispelled by a study of the microscopic specimens which accompany this thesis. I re-insist here upon the difficulty and complexity of the problems which are met with in considering the subject of subdural membrane formation for two reasons. In the first place it serves to explain to some extent why so many important microscopic changes which occur were not described in the recent publication on the subject for which I was jointly responsible; and in the second place it perhaps atones in some measure for my inability to explain the meaning of many of the

morbid appearances which I have described in the foregoing series of cases.

With a view to the attainment as far as possible of clearness and conciseness I shall divide my remarks under this heading into the following three sub-sections.

I. The naked-eye anatomy of subdural membranes and allied morbid conditions.

II. The morbid appearances which may be observed in superficial horizontal sections of the dura mater.

III. The morbid processes concerned in subdural membrane formation.

I. THE NAKED-EYE ANATOMY OF SUBDURAL MEMBRANES AND ALLIED CONDITIONS.

Out of 148 post mortem examinations which I have performed at the Royal Edinburgh Asylum there have been 34 cases in which there was a separable false membrane on the inner surface of the dura. (23 per cent). Four of these false membranes were the result of a large recent haemorrhage into the subdural space of which the source was ascertained. In the case of three of them it was from a pial vessel, and in the fourth it was the result of an intracerebral haemorrhage bursting into the subdural space. In two cases haemorrhagic false membranes were found in the cerebellar fossa accompanying more extensive subdural membranes. Only two were of the cystic form. In seven cases the false membranes consisted merely of red blood clots adhering to the surface of the dura, without any indication of haemorrhage having occurred from a pial vessel. In addition to these 34 cases in which there were subdural membranes there were 21 in which were recognised morbid conditions which I believe to represent early stages in the process of subdural membrane formation. The morbid appearance in 15 of these consisted in the presence of granulations on the inner surface of the dura. In four

it was a yellow or rusty staining, usually accompanied by granulations. In the remaining two it consisted in an opaque silvery appearance over extensive areas of the internal dural surface. These 21 cases all occurred with a few exceptions among my last 70 cases, and it is certain that in the preceding cases many morbid appearances were overlooked which I now recognise and place in this category. Taking my last 50 cases alone I find that it is the exception to see a dura free from one or other of these morbid changes in a patient dying insane.

I have nothing of importance to add in this subsection to the descriptions of the naked-eye appearances of subdural membranes given in the review of previous literature (Page 25), but it is necessary to say something further here about those other morbid appearances recognisable on post mortem examination which I maintain represent an early stage of the same morbid process. My observations lead me to the conclusion that we must divide subdural membranes into two classes. On the one hand there are those which are clearly the result of a large haemorrhage into the subdural space, the source of which in the recent state can usually be recognised. On the other hand there are those which develop as the outcome of a widespread morbid process occurring in the dura itself, the initial

stages of which cannot be recognised with the unaided eye. This I maintain is the true subdural membrane formation of the insane. It is I believe a disease which, except in chronic drunkards is rare in the mentally sound, while the former class of cases is probably as common in those dying mentally sound as in those dying insane. The earliest indications appreciable to the unaided eye of the morbid process which leads to the development of the true subdural membranes of the insane are one or other of the three appearances already mentioned. Granulations are exceedingly common. They appear as minute asperities on the inner surface of the dura. When present they are usually specially numerous and large over the body and lesser wings of the sphenoid, and upper surface of the tentorium cerebelli, but are often easily recognisable over the whole of the inner surface. They are specially common in senile insanity Yellow or rusty staining is due to the presence of haematoidin granules near the surface. They require to be present in considerable quantity to render their recognition possible with the unaided eye. Many duras from the insane which appear at the post mortem free from any pigmentary deposit are found on microscopic examination to contain haematoidin granules in abundance near the inner surface. The opaque

silvery change in the inner aspect of the dura is of much interest on account especially of the light it probably throws upon a very difficult question connected with the development of concentric bodies. I have only observed it in two cases, both of which are contained in the foregoing series. (Cases No. 25 and 26), but in two other cases (No. 27 and 38) I have recognised on microscopic examination the occurrence of the same morbid change which gives rise to it. This is a degenerative change in the endothelial cells of the surface and of the perivascular canals, to which the term "vitreous degeneration" might possibly be suitably applied. It will be further described presently. In case No. 25 this morbid appearance extended over practically the entire inner surface of the dura. It is an appearance which it is exceedingly difficult to describe in words, and those which I have used for the purpose may convey little impression as to its nature. It reminds one very much of the appearance presented by the under surface of the leaf of the potentilla anserina, though it is of a much less glistening character.

There are some points regarding the naked-eye anatomy of delicate fibrinous membranes to which I wish to allude, but their considerations must be deferred until certain microscopic characters which they present are described.

II. THE MORBID APPEARANCES WHICH MAY BE OBSERVED IN SUPERFICIAL HORIZONTAL SECTIONS OF THE DURA MATER.

1. Morbid changes in the Endothelial Elements of the Dura.

These changes are both proliferative and degenerative. They are of a very marked character and are, I believe, of great importance in relation to subdural membrane formation.

Out of the 43 cases of which I have described the microscopic appearances of the dura, in 25 there was distinct evidence of proliferation of the surface endothelium. (see for example specimens No.64 and 120.) (Fig.25.) In the same number of cases, though not in the identical ones, there was unmistakable evidence of proliferation of the endothelial cells which form the walls of the perivascular canals. As a rule the condition when present in the one situation is also found in the other. Localised aggregationsof proliferated surface endothelial cells constitute one form of granulation. The cellular aggregations which occur in the perivascular canals are probably identical with those which have been observed near the surface in transverse sections by the advocates of the inflammatory theory, and which have been regarded by them as

constituting a small round celled infiltration. For my own part I am perfectly confident that I am accurate in stating that these cellular aggregations are in almost every instance due to proliferation of the endothelial cells of the perivascular canals. In superficial horizontal sections deeply stained with haematoxylin their endothelial character is perfectly evident. (Fig.14.) The following specimens are submitted in support of this intention, - No. 65, 70, 129 and 77. In the last specimen at the spot marked there is seen a vessel in the perivascular canal of which there are cells which are certainly leucocytes. The contrast between these and the cells which occur in the perivascular canals at the other spots to which attention is directed will be seen to be quite distinct.

The degenerative changes which occur in these endothelial cells, especially in association with their proliferation, are of several varieties. I cannot claim to have thoroughly worked them out as yet, and will only attempt to indicate some of the more important morbid appearances which may be observed.

The cell-plate frequently undergoes a granular and disintegrative change. In the normal condition this portion of the cell can seldom be distinguished in haematoxylin and eosine preparations.

But in this morbid state it stains to some extent with eosine and its outline can therefore frequently be distinguished. The granules often take the eosine stain somewhat deeply. These disintegratory cell-plates may be seen on the inner surface in specimens No. 66, and 192. (Figs. 24 and 31.) The same change also occurs in the endothelial cells of the perivascular canals, but in them it is more difficult to recognise. It probably always accompanies the more prominent degenerative changes described below which specially affect the nucleus.

A common and important degenerative change in these endothelial cells is one which begins as a vacuolation of the nucleus. (Figs. 30 and 27.) This I believe is the early condition of the vitreous degeneration which is described in cases 25, 26, 27 and 38, and which I have recently also recognised in specimens from other cases, - more particularly in No. 16. (see specimen No. 96) Numerous clear areas develop within the nucleus, gradually filling it and swelling it out, until an inflated-looking glassy or vitreous body is produced. (Fig. 28. Micro. spec. 37 and 38) The large pale blue cells (Fig.29) so frequently seen in haematoxylin and eosine preparations of subdural membranes, as well as in superficial horizontal sections, are probably products of the same degenerative process. The extreme degrees of this vitreous degeneration may be seen in

in specimens from the above enumerated cases, e.g. Nos. 150, 157, 178, 193, 198, 96 and 149. It affects large areas of the surface endothelium and also that of the perivascular canals. The clear, crystalline or vitreous substance which is produced by this change does not stain with eosine or haematoxylin, and it is not blackened by osmic acid. On the surface it frequently forms large crystalline-looking masses. (Micro. Spec. No 159) When extensive continuous areas of the endothelium of the perivascular canals are affected the enclosed vessel becomes compressed and its lumen obliterated. (Specimen No. 150)

As the result of a further change the individual globules of this degenerative material coalesce, and at the same time become somewhat opaque and assume a strong affinity for eosine. By a still further change it may develop into concentric bodies.

Another degenerative change in the nucleus of these endothelial cells which is probably closely related to that just described, is one which leads to the formation of concentric bodies of the starch grain form. The whole subject of concentric bodies is considered under a special heading, and I shall not here pursue it further.

2. Phenomena following effusion of blood upon the internal surface of the dura.

These phenomena may be studied to great advantage in superficial horizontal sections of the dura, and as there seen are of great interest, not merely because of the light which they throw upon the pathology of subdural membrane formation, but also from the point of view of general pathology.

In at least 26 of the cases I have described there may be observed on the dural surface delicate granular and fibrillated shreds or films which I think it may be proved are all mainly composed of coagulated fibrin. Sometimes they are, no doubt, exceedingly difficult to distinguish from a subendothelial layer of loose connective tissue when this exists. Examples of this difficulty are seen in specimen No. 12† and 187‡. I maintain that this coagulated fibrin is a blood-clot and that it is not an inflammatory exudation. The grounds for this opinion will be fully discussed presently. In the meantime I wish merely to trace the changes which take place in these fibrinous films and in the subjacent tissues in the course of their reaction towards them.

When a small effusion of blood takes place into a subdural space in which there is no excess of

fluid, a thin layer of red and white blood-clot forms on the inner surface of the dura in the neighbourhood of the point from which the blood escaped, - the red clot occupying the centre and the white clot spreading out as a delicate surrounding film which generally extends much further than is appreciable to the unaided eye. Typical examples of such false membranes occurred in cases 29 and 30. (See also Micro. Spec. 74) The red corpuscles begin to break up with remarkable rapidity into granular débris. This is shown inmicro.specimen No. 135, from case 23 in which to judge from the histroy the haemorrhage began only seven hours before death. That this disintegration is not a mere post mortem one is proved by the fact that it can be seen that the leucocytes have been taking up the granular débris into their interior, the protoplasm of many of them being filled with it. The fibrin threads are at first very distinct and are spread out on the surface of the dura in the form of an even layer such as is seen in specimens No. 41 and 91. Very soon, however, they become less evident and the coagulum assumes under the microscope a granular appearance, part of which, however, may be due to the products of the disintegration of leucocytes and also of red blood corpuscles , which it can be shown are always present to some extent even in the white portion of

the clot in the recent state. A continuous sheet or film thus becomes formed which often presents little trace of the original threads of which it consisted. (See for example specimen 65 at the ~~object~~ spot marked ", and 128.) Frequently at an even earlier stage than this the clot shows signs of undergoing contraction. This leads to the formation of openings in the film so that a fenestrated appearance is produced, (Fig. 55) such as is seen in specimens No.176 and 40. Essentially the same appearance is seen in specimen 86 which gives a horizontal view of a false membrane composed of fibrin in process of becoming vascularised. It will be seen that the coagulum has assumed the form of many inaccurately superimposed, partially united, flattened networks or fenestrated membranes. Specimen 89 from the same case, which is a superficial horizontal section of the dura made after the removal of the false membrane, shows that the shreds of the latter which remain adherent to the dura have the same appearance as that which is produced by a minute effusion on to the surface. (See also specimen 117.) The contraction continues until the fenestrated sheet becomes a network. (Fig. 54. Micro. Spec. 89) The cords of this network have usually a longitudinally striated appearance and often stain very deeply with eosine, and sometimes also with haematoxylin.

(Fig. 54. See specimen 197). They tend to split across and then to further contract and curl up. (See specimen 57 at one of the spots marked + and also 79). Before these changes have proceeded thus far the fibrinous coagulum usually begins to be penetrated by new capillaries which shoot out from the superficial vessels of the dura. A typical example of this process is seen in specimen No. 202 at the spot marked + . Specimen 86, already referred to, shows the same process on a much more extensive scale. This outgrowth of vessels may begin within a few hours of the occurence of the blood-effusion. This at least seems proved by case 23, as in specimens 136 and 139 new capillaries can be seen penetrating the clot of blood, which, judging from the history began to be effused only seven hours before death. Another effect upon the dural tissues of the presence of a fibrinous film seems to be rapid proliferation of the surface endothelial cells,- a condition which, however, is often already established to a marked degree in those cases in which haemorrhage from the superficial dural vessels is specially liable to occur shortly before death. Many of these proliferated endothelial cells pass into the false membrane. (See specimens 74 and 137). Under certain circumstances, probably especially in the case of thin fibrinous films, a large number of the

new capillaries become obliterated. (Fig. 59). This is seen in specimen 202 and 162. Why this obliteration should occur is difficult to explain. Many of the appearances suggest that it may arise from compression of the new vessels by the contracting fibrin which can be seen to envelop them with a sheath. (Figs. 56 and 57. Micro. spec. 203). These obliterated capillaries, which frequently assume a homogeneous hyaline appearance, occur very frequently in superficial horizontal sections, often presenting little or no trace of the clot which caused their development. They may be lying on the surface of the endothelium or this may have grown over them. Though I have previously committed myself to an opposite opinion (156), I am now inclined to think that all capillaries presenting this change are new vessels and that the original capillaries of the dura never become affected in a similar way. Appearances are, nevertheless, occasionally seen (as in specimen No. 199 at the spot marked +) which point to the possibility of the original capillaries of the dura being compressed in a similar way by fibrin which has coagulated within the perivascular canal. That contracting fibrin can cause obliteration of capillaries is, however, merely a hypothesis, and I do not here commit myself to the view that such

a process occurs. Some of the changes which take place subsequently to the above are exceedingly difficult to follow. What becomes of the cords of the contracted coagulum I have been unable to determine. The obliterated capillaries become covered over by endotheliam, and probably ultimately develop into fibrous cords. When the new capillaries do not become obliterated they naturally form the vessels of the granulation tissue which develops on the surface of the dura, replacing the blood-clot and forming an organised false membrane.

3. Morbid Changes in the Superficial Vessels of the Dura.

A common change is one which consists in a slight thickening and granularity of the capillary wall, which at the same time stains abnormally deeply with eosine. (Figs. 15 and 16. Micro, Spec. 83, 80 and 132) Associated with this change there are frequently found, specially in senile insanity, haematoidin granules lying outside of the vessels, either in their perivascular canals or in the inter-vascular areas but still subjacent to the surface endothelium. (Fig. 19, Micro. Spec. 70, 79, 84, 90, and 121) The proliferation of the endothelial cells of the perivascular canals which has been already described is always accompanied by some degree of this thickening and granularity of the capillary wall, though the latter may be seen in the absence of the former. These proliferated endothelial cells beyond all question frequently compress the capillaries interfering partially or completely with the passage of blood through their lumen. The vitreous degeneration which may affect the endothelial cells of the perivascular canals, as previously stated may also lead to the obliteration by compression of the lumen of the contained capillaries. Fatty changes are found to some extent in the superficial capil-

laries, especially in those which already show evidence of morbid change in thickening and granularity of their walls and proliferation of the endothelial cells of their perivascular canals. These fatty changes may be seen in specimen No. 46, which is immediately from a case in which there were numerous ante-mortem haemorrhages. (See also specimen No. 63 at the spot marked ###) Large masses which blacken with osmic acid are sometimes found within superficial capillaries, and probably arise from coalescence of fatty globules which have arisen in the capillary walls. (Fig. 27. Spec. 46.) Similar masses are sometimes seen outside of the vessels. (Specimen 161) They probably arise from the endothelial cells of the surface and of the perivascular canals which often contain numerous fat granules. Recent capillary haemorrhages are very frequently found to have occurred on to the surface of the dura in association with any one or other of the above mentioned degenerative changes in the walls of the vessels or of their perivascular canals. When very minute these blood effusions may be entirely sub-endothelial, often being merely into the perivascular canal. Small surface haemorrhages may be evidenced only by the presence on the surface of the dura of the granular and fibrillated films and networks already described.

The source of the haemorrhage can, however,

frequently be observed. See the following specimens, 39, 43, 62, 69, 99, and 182. (Fig. 22.) An example of a quite microscopic haemorrhage from a capillary which has been obstructed by localised proliferation of the perivascular endothelial cells is seen in specimen No. 45 at the spot marked. (Fig. 21.) Recent haemorrhages are also sometimes seen from the deeper vessels of the dura. Examples are seen in Specimens 54, 55 and 85. Great distension of superficial capillaries is frequently observed, specially in cases in which there are recent haemorrhages Such vessels are often very difficult to distinguish from new capillaries owing to the perivascular canals being obscured by the dilatation. (Fig. 13. Specimen 42.) Fibrin threads can frequently be observed within capillaries both of false membranes and of the dura. See for Example specimen 129 at the spot marked. New capillaries may frequently be seen in process of development on the surface of the dura over degenerated and occluded vessels. (See specimen 165.) These new capillaries may spread over the surface without the previous occurrence of haemorrhage. (See specimen No. 166) (Fig. 5) New capillaries may also frequently be seen to develop in the perivascular canals within which the

original vessels have become occluded. See specimens 158 and 164. Haemorrhages from these new vessels are exceedingly common (Fig. 12.) and haematoidin granules often occur in abundance in their vicinity.

4. Hyaline Changes.

In the descriptions of the microscopic characters presented by the series of cases, I have used the term "hyaline" merely in a general descriptive sense, implying a material which has a homogeneous glassy appearance, and which stains deeply with eosine in haematoxylin and eosine preparations but gives no waxy reaction. Irregular masses, cords, rods, networks, and fragments of a substance of this kind may be observed very frequently in superficial horizontal sections of the dura as well as in many subdural membranes. (Figs. 36, 37, 39 and 43.) The origin of many of these is difficult in the extreme to trace. Some of the appearances presented are most conflicting. It would be endless to enter into a discussion of all the various possibilities which they suggest, and I shall confine myself to a brief statement of the present position of my own views as to their interpretation, which are still far from being settled. The subject is one which will entail much additional labour before it can be fully worked out.

The cords of the networks formed by contracting fibrinous films on the surface of the dura often assume a hyaline appearance, staining deeply with eosine. At the same time they are usually slightly longitudinally striated. (Specimen No.171). Capillaries which have penetrated a fibrinous false

membrane, and have afterwards become obliterated in the manner already described, frequently show a hyaline sheath which is composed of altered fibrin. (Specimen 203). At a later stage these vessels assume the appearance of homogeneous hyaline rods (Fig.56), which become covered over by the surface endothelium. This is probably the nature of the hyaline rods in specimen No.7, which can be traced to continuity with original dural vessels. The subendothelial position of some of these obliterated capillaries can be distinctly seen in specimen No. 101, which is a silver preparation. In specimen No.102, similar vessels can be seen superficial to the endothelium at the spot marked.

Certain dense fibrous tissue strands, which in some parts of the dura lie just below the surface endothelium, may assume a similar homogeneous hyaline appearance. See for example specimen No.189 at the spot marked. The hyaline rods which can occasionally be seen to lie on each side of a superficial dural vessel also probably arise from a peculiar change in the fibrous tissue outside the perivascular canal wall. (See figs.17 and 18, and specimens 105 and 167).

The substance which arises from vitreous degeneration of the endothelial cells undergoes a further change, by which it assumes the staining reactions of

hyaline material. It may appear as irregular masses on the surface or as long rods which fill the perivascular canals. (See specimens for case No.25). The hyaline cords seen in specimens 159 and 160 may also have had this origin.

A typical example of the difficulty that there frequently is in deciding upon the source of these hyaline rods is seen in specimen No.195 at the spot marked, where the diagnosis between an obliterated vessel and a modified strand of fibrous tissue is practically impossible.

5. Concentric Bodies.

I distinguish two forms of these structures. There is first the typical form which is usually of large size, often being three or four times the diameter of a normal superficial dural capillary, but which is specially distinguished by its rounded shape and hyaline appearance. Its affinity for eosine is usually very strong. The concentric markings, which are merely thin dark lines, may be few or numerous. Surrounding these concentric bodies there is usually a more lightly stained, sometimes homogeneous, sometimes distinctly fibrous and laminated, broad or narrow capsule. (Figs.47 and 49. Specimens 14, 77, 104, 127). They are seen in transverse section in specimen 17. The second variety is that which I have frequently referred to as "concentric bodies of the starch grain form." They are much smaller than the hyaline variety, though also varying considerably in size, have usually an oval shape (which is also assumed by their concentric markings), and in haematoxylin and eosine preparations stain of a voilet colour and appear somewhat opaque. Very rarely, however, a portion of their structure may be seen to have hyaline characters. Their appearance suggests at once the simile of a starch grain. (Figs.50 and 51. Micro. Spec. 82). Concentric bodies of the large hyaline form are much the more common. I have

found them in 26 out of the 43 cases here described. In some cases they occur in very large numbers. They are by no means confined to the insane, though they certainly occur in such patients in far greater abundance than in the mentally sound, as far as I have seen. They were found in the following general hospital cases, - Nos. 31, 38, 39, 40, 42 and 43. They may be found in duras which show practically no other morbid change. They are certainly most common in old people, but probably may occur at any age. In cases 31 and 38 the patients' ages were nineteen and twenty-six respectively, while several of the asylum patients in whose duras they were found were under 30. Concentric bodies also occur, often in very large numbers, on the surface of the arachnoid, especially in association with the milky change which is so common in the insane. In this situation they were evidently observed by L. Meyer in 1862 (Virchow's Archiv. - Band XVII.), and their occurrence is also mentioned by Obersteiner (141). A description of them as they appear in the arachnoid will be found in the Edinburgh Medical Journal for May of this year in a paper by Dr Middlemass and myself on "Morbid Conditions of the Pia-Arachnoid." They also occur in the Pacchionian bodies, an observation which has also been made by Obersteiner. They are seen in this situation in specimen 55. The

following are some of the reactions of these hyaline concentric bodies. They stain deeply with carmine and haematoxylin, very readily parting, however, with the latter when subsequently treated with hydrochloric alcohol. They stain deeply with eosine (showing a much greater affinity for it than for haematoxylin) and with many of the anilin dyes, such as acid fuchsine (specimen 73), saffranine and methyl violet. They are not blackened by osmic acid (Specimen 63). They assume a dark, brown colour in silver preparations (Fig.48. Specimen 97). They do not give a waxy reaction with methyl violet. They are not readily affected by the action of aqueous solutions of mineral acids (Specimen 87). The statement of Obersteiner that they consist of phosphate and carbonate of lime is therefore clearly an error, and consequently the term "corpora arenacea" seems an unsuitable one for them. I have never seen any evidence of retrogressive or disintegrative changes in them. I have as yet no observations to record as to their relations to "amyloid bodies" and concentric bodies of the prostate.

I have expended much labour in an endeavour to determine the method of development of these concentric bodies, but they still keep at least a large part of their secret. The origin of those of the starch grain form is evident enough. They arise

directly from the nucleus of a single endothelial cell, as the result of a peculiar degenerative process. They are not developed merely from the surface endothelial cells, though this is their most common source. I have frequently observed them in the perivascular canals, sometimes even deep in the dura. (See specimen 68). The development of the hyaline concentric body is a much more difficult problem. One is much helped, however, by a study of their formation on the surface of the arachnoid, where there are no hyaline rods to complicate the appearances. As will be found described in the paper just referred to, they arise on the surface of the arachnoid as the result of a coalescence of certain of the endothelial cells which have undergone a peculiar degenerative change. This change, I am now satisfied, is the same as that which I have already described as vitreous degeneration. This seems clearly established by the microscopic specimens from case No.25, in which it can be seen that the crystalline or vitreous masses in the perivascular canals coalesce and develop into a rod which stains deeply with eosine, and subsequently breaks into portions which change into concentric bodies. (See specimens 150 to 155). It is easy to understand that the endothelial cells of the surface of the dura mater, which undergo a similar change, may

develop in the same way into concentric bodies. It may, indeed, be accepted as practically certain that this is one way in which they are formed. This is confirmed by the fact that they give in silver preparations the same reaction as the surface endothelial cells. A developmental form of this kind is probably represented by the appearance in specimen 65 at the spot marked. (See also Fig.44). Some other interesting developmental forms, pointing to a similar origin, either from the surface endothelium or from that of the perivascular canals, are seen in specimens No.67, 111, 112, 114, 125 and 127. (Fig. 45). It may be that their origin in every case is from this vitreous degeneration of the endothelial cells, but there are many appearances which are exceedingly difficult to explain upon this hypothesis. Many of the hyaline rods already described show what appear to be transition forms in the development of concentric bodies. To explain some of these appearances I must confess myself in the meantime quite at a loss. I shall not attempt here to discuss all the possibilities which they suggest. I shall merely say that many of the hyaline rods which appear to develop into concentric bodies are exactly similar to others which may be traced into continuity with vessels, with dense strands of fibrous tissue, and even with fibrinous films on the surface of the dura, so

that it seems out of the question to contend that they must all have arisen from vitreous degeneration of the endothelium of the perivascular canals. Some of these transition forms are seen in specimens 9, 11, 14, 91, 105, and 114. (Figs.40, 41, 42, 43 and 46). Another exceedingly interesting specimen is No.110 at the spots marked. (Figs.37 and 43). If any proof were required that these concentric bodies are developed in the dura and that they have not merely been shed from the arachnoid surface, it is furnished by specimen 108, in which, deep in an inflection of the spinal membrane, two concentric bodies can be seen to have formed.

6. Mulberry bodies.

I have applied this name to certain curious structures, which may occasionally be seen on the surface of the dura in superficial horizontal sections. They consist of masses of fairly large, oval or rounded cells of a homogeneous appearance, which stain of a deep purple colour in haematoxylin and eosine proparations. They have usually a faintly stained gelatinous capsule. Single cells of the same appearance may occasionally be seen, as well as small groups of two or more. I have been unable to trace their origin. They are seen in specimens 71+ and 79+, and are illustrated in figures 52 and 53.

7. Granular patches in the fibrous tissue.

In superficial horizontal sections stained with haematoxylin and eosine, or with alum carmine and eosine, there may frequently be seen scattered over the surface numerous small irregular patches, which stain deeply with both dyes and have a faintly granular appearance. (Fig.60). They are seen in specimens 39+ and 184+. They may frequently be observed also at a deeper level in horizontal or transverse sections. They are exceedingly common in duras from general hospital patients as well as in those from the insane. Probably a further development of the same change is seen in the large degenerated areas described in case No.1. (Specimen 15. Fig. 61). As these were fully described under that case, and as I have met with no other instance of their occurrence, I shall not enter into their microscopic characters in this section. A connecting link between the granular patches and this more extensive change is probably seen in specimen No.109 at the spot marked+.

Another connective tissue change, which may be mentioned here, is one which often affects the corpuscles in duras which present other evidences of a morbid condition. It may be seen in specimen No. 205 in association with a false membrane. The connective tissue cells are much swollen and their protoplasm is granular and stains deeply with eosine.

8. Granulations.

Of these there may be distinguished three varieties. In the first place, there are some which consist almost entirely of proliferated endothelial cells (Specimen 103), corresponding exactly to the structure of the arachnoid granulations on the opposite wall of the subdural space. (See paper in Edinburgh Medical Journal for May of this year already referred to.) The second variety is developed from this form. The proliferated endothelial cells are specially liable to develop into concentric bodies, groups of which are frequently to be observed among them. (Specimen 112). All the surrounding cells may disappear, leaving the more stable concentric bodies adherent to the surface and projecting from it. Many of the granulations recognisable with the unaided eye are found on microscopic examination to consist solely of groups of these concentric bodies. (Specimen 104). There is probably also a third form which is composed of true granulation tissue developed around new vessels, which have shot out upon the surface of the dura, subsequent to the obliteration of subjacent capillaries. It is covered by endothelium. This was described as the typical granulation in the recent paper by Dr Middlemass and myself (156) before the importance of the endothelial changes which occur was fully recognised.

III. The Morbid Processes concerned in Subdural Membrane Formation.

The researches of which I have endeavoured to give a systematic account in the foregoing pages lead me to the conclusion that in considering the morbid processes concerned in subdural membrane formation we must at the outset distinguish between two classes of cases which occur. In the first place there is a class in which subdural membranes develop as the direct outcome of a widespread morbid process occurring in the tissues of the dura itself, and in the second place there is a class in which they are formed as the result of a haemorrhage into the subdural space which takes place quite independently of this morbid process in the dura. In the first class of cases we see the true subdural membrane formation of the insane, a disease which, while it is very common in them, is comparatively rare in the mentally sound. On the other hand in the second class we have a morbid condition which is probably as common in the mentally sound as in the insane.

It will be most convenient to consider first the morbid process concerned in the development of subdural membranes which are to be included in the latter class. The causes of the haemorrhage are merely those which give rise to intracranial

haemorrhage in general and present no special feature in this relation. Its source is probably most frequently a pial vein, but may also be from a pial artery, a venous sinus; an artery at the base, an intra-cerebral vessel or one near the inner surface of the dura. Taking the example of a fairly large haemorrhage, one of the first effects of the presence of the blood in the subdural space is to set up very active proliferation of the cells of the endothelium which lines the subdural cavity. Many of the proliferated cells become shed and pass into the effusion, in which they may afterwards be recognised in large numbers. (Specimen 106). After a certain interval coagulation of the blood takes place. This commences on the walls of the subdural space so that a white clot forms at the periphery and envelops the red clot which forms later. The peripheral white clot, probably mainly owing to the effects of pressure, often forms quite a dense membrane. (Case 23) In the case of small haemorrhages the clot is spread out as a thin layer, the red portion occupying the central area and the white appearing as a delicate surrounding film which gradually becomes more and more attenuated until it is lost to view on an apparently healthy surface. The microscope reveals the fact that it extends much

further than the unaided eye can trace it. (Case 29) If, as occasionally occurs, especially in chronic insanity, there is marked excess of fluid in the subdural space the effused blood becomes mixed with it, and after a time its fibrin coagulates out upon the walls of the subdural cavity, often forming a film which extends over the entire surface. (Case 18) At first the clot is not adherent to either wall. Very soon, however, new capillaries shoot out into it from the dura so that vascular connections are formed by which it adheres to the outer wall of the space. Vessels very seldom penetrate it from the inner wall for the reason that the arachnoid is a practically non-vascular structure. The arachnoid aspect of the clot becomes covered over with a layer of endotheleal cells which are continuous with those of the surface of the dura at its margin. (Greenfield, 93). (See microscopic specimen No. 172.) Thus the false membrane does not lead to the obliteration of any portion of the subdural cavity, but forms for it a new outer wall. The subsequent stages are practically identical with those of organisation of a thrombus in a vein, the clot in time becoming entirely replaced by granulation tissue. In very large haemorrhages the red clot in the centre is apt to break down before

the process of organisation has reached it, and in this way a typical "arachnoid cyst" is produced. After false membranes formed by comparatively small haemorrhages have become organised they closely resemble those which are formed by the second morbid process presently to be described, and the further changes which may occur in them are also probably very similar.

Coming now to what I regard as the true subdural membrane formation of the insane, we have a process to consider which is in certain respects essentially the same as the preceding, but in others entirely different. It is the same in that a part of it consists in the occurrence of a haemorrhage into the subdural space and all its consequences. It is different in that it consists in the occurrence of a Multitude of haemorrhages, in that these are due to a widespread definite morbid condition of the tissues of the dura, and in that there is a part of the process by which the false membrane may be formed in which haemorrhage plays no part. I would have it clearly understood, however, that there is no sharp distinction which can be made between the two classes. They merge into each other, and there are many cases which cannot be properly assigned wholly to either group. Yet I think it will be evident presently that the classification is a scientific and necessary one.

Before sketching this process, however, I must diverge for a moment to direct attention to a matter which to my view has very important bearings upon the pathology of subdural membrane formation in the insane, but which does not seem to have been grasped by writers on the subject. I think that it can be shown that with a few exceptions all the false membranes found in the subdural space of the insane are the development of a few days preceding death. Taking the cases that I have described, the only one in which the microscopic characters of the false membrane point to its having been of several weeks standing is No. 24. Of the other cases from the insane in which there was a false membrane present the most highly organised was that which occurred in No. 2, and I think that a study of the case renders it very probable that, though there certainly were advanced degenerative changes in the dura, the false membrane was mainly the result of a somewhat large haemorrhage which occurred during the congestive attack a fortnight before death. Of the eleven remaining cases from the insane in which there was a false membrane recognisable to the unaided eye the only one in which it is composed of fully organised tissue is No. 5. All the others show a basis of fibrin threads which points to a recent origin.

To these ten cases which must be regarded as of recent development, there have to be added nine others in which there was microscopic evidence of the presence of false membranes of a fibrinous structure. Further, the accounts given of the naked-eye appearances of subdural membranes by various writers (e.g.- Wiglesworth, 134) are in most instances clearly descriptions of membranes mainly of a fibrinous structure. It seems to me therefore beyond all question that we must regard the great majority of the false membranes which occur in the subdural space of the insane as forming in the course of a few days or a few hours preceding death. To put it in another way, in each of these nineteen cases a fortnight before death, whatever morbid changes there might have been in the actual structure of the dura, there was no false membrane in the subdural space. In the general hospital cases examined, putting aside five in which there was head injury, it was found that in seven out of the remaining ten there was either naked-eye or microscopic evidence of haemorrhage (often exceedingly slight) having occurred from the vessels of the dura very shortly before death. Therefore I think we must further recognise that there are certain factors furnished by the moribund condition which predispose to haemorrhage from the dura in all patients. These

factors are, I believe, merely the fatty and other more subtle molecular changes which occur in the walls of the dural vessels in common with many other tissues of the body, to some extent in the last stages of practically all diseases not resulting in sudden death, but especially in acute illnesses attended with high temperatures and in the last stages of chronic diseases towards the termination of which there is prolonged and great bodily enfeeblement. At the same time we have to account for the undoubted fact that subdural membranes appreciable to the unaided eye are far more frequently met with at post mortem examinations upon the insane than at those upon general hospital patients. From the investigations which I have made I conclude that this is due to the circumstance that the insane are specially prone to a chronic morbid condition of the dura on account of which these same pathological factors which cause capillary, and often larger, haemorrhages from the dura in the mentally sound shortly before death, make their influence felt at a much earlier stage and to a much more marked degree.

It is a fact familiar to all those who are accustomed to see post mortem examinations performed upon asylum patients that the pia-arachnoid is in the majority of the insane found to be affected by a morbid condition which is evidenced to the naked eye in marked thickening and milkiness especially over the convexity of the brain. These morbid appearances are comparatively rare in general hospital patients occurring in them chiefly in cases of chronic alcoholism, and to a slight degree as a common senile change. In two papers upon this disease of the pia-arachnoid as it occurs in the insane, recently published by Dr Middlemass and myself (Edinburgh Medical Journal, April and May, 1895,) we have given an account of a research that we have made into its pathology, and we have come to the conclusion, which I think will be accepted by anyone who will take the trouble to examine the microscopic evidence, that the condition is not an inflammatory one as has generally been held, but that it is a chronic degenerative one attended by marked proliferative and degenerative changes in the endothelial cells that line the outer surface of the membrane and the subjacent arachnoid spaces, and also by an extremely slow hyperplasia of the connective tissues. Now I have to make the statement, which involves a highly

important generalisation, that on the opposite side of the subdural cavity there occurs in a like proportion of the insane a morbid change of an exactly analogous kind. The corresponding anatomical elements are the endothelium of the inner surface of the dura and the perivascular canals, which, like the arachnoid spaces, are lined by endothelial cells. The microscopic investigations of which I have given an account prove beyond all question that in both of these situations the same proliferative and degenerative endothelial changes occur as in the corresponding situations in pia-arachnoid. In relation to the formation of subdural membranes in the insane the most important of these morbid changes in the dura is the proliferation of the endothelial cells of the perivascular canal walls. This proliferation indicates a morbid nutritional state which must at the same time affect the capillary walls and cause them to be weakened. In them the indications of disease are not so distinct, but still it is evidenced by the thickening and granularity already described. This in itself must cause weakness of the walls of the capillaries and render them less able to resist the normal blood-pressure. But there are other important changes which predispose to haemorrhage from these

vessels. The proliferated endothelial cells in a perivascular canal may compress the contained capillary and partially or completely obstruct it. The already weakened capillary dilates behind the obstruction and may rupture. Another result which may follow complete obstruction of a capillary is the development of a new one in the same perivascular canal. New capillaries are always weak so that here again is another danger of haemorrhage. But when the superficial capillaries become occluded the new capillaries which form do not always confine themselves to the perivascular canals. These indeed may be blocked by endothelial cells, blood corpuscles and haematoidin granules, so that capillaries cannot easily push their way along them. Therefore they grow out upon the free surface of the dura, often forming quite a leash or network of dilated vessels. (Specimen 166) They are accompanied by fibroblasts along with which they may quickly develop an area of granulation tissue, forming a microscopic organised false membrane. It will be seen that by such a process a subdural membrane may be formed independently of the occurrence of haemorrhage. This is probably, however, a process which seldom occurs to any very great extent unaccompanied by haemorrhage from the new

capillaries, which like other new capillaries are extremely prone to bleed. It is further to be pointed out that the flow of lymph along the perivascular canals must be greatly interfered with by the morbid products which accumulate in them. Now whatever the exact functions of the perivascular canals may be, it is easy to understand that this obstruction of them must in itself produce an abnormal condition of matters which will be an additional source of weakness to the contained capillary. All these facts regarding the structural changes which occur have been pointed out in the preceding pages and are demonstrated by microscopic specimens. The blocking of the perivascular canals and compression of the capillaries by the vitreous change in the endothelial cells of the former is also to be remembered. Its exact relation to subdural membrane formation, however, is in my own mind still a matter of uncertainty. I am inclined to think that the extreme degrees of it seen in some of the cases I have described must have been established only a very short time before death. There is satisfactory evidence, however, that these proliferative and degenerative changes in the dura of the insane are not the mere development of the immediately antemortem period. In case No. 10, in which death occurred from drowning at a time when the patient

was not in a worse state physically than he had been in for months previously, the proliferative changes at least were present, and there is also evidence of the occurrence of old capillary haemorrhages in the presence of haematoidin granules. The large quantities of concentric bodies, as well as of hyaline material in other forms, which occur in the dura of many of the insane is evidence of the long standing character of the purely degenerative changes; and further, though slight proliferation of the endothelial cells was frequently seen in the duras I examined from general hospital patients, it almost never occurred to any corresponding degree to that in which it was found in the insane, and haematoidin granules never occurred in connection with it. So long as the patient is in good physical condition the tendency to haemorrhage from these morbid dural vessels is probably not very great. Nevertheless a certain number of subdural membranes which bear evidence of having been in existence for comparatively long periods, - of which many examples have been described by various writers, - are, I think, clearly to be attributed purely to the effects of this degenerative process in the dura. I have not myself yet met with a single case in the course of 148 post mortems on the insane in which the microscropic and other evidence pointed to the false

membrane having had origin more than a fortnight before death. (No. 24 was not one of my own cases) That small haemorrhages do from time to time occur from these vessels without the addition of any new morbid factor is, I think, proved, though not quite conclusively, by the frequent presence of haematoidin granules around them. When, however, such patients are attacked by any severe bodily disease which proves fatal, the fatty (Specimen 46.) and other molecular tissue changes which occur especially towards the termination of the illness, affect these already weakened dural vessels in common with others throughout the body. It may be that such molecular changes affect them much more severely than they affect vessels which were previously healthy, and even that the degenerative processes already established are given an additional impulse by the general nutritional failure. But whether these possibilities are so or not it is easy to understand that in the case of such already weakened vessels the period at which they become unable to withstand the normal pressure of the blood will occur much earlier than in the case of previously healthy vessels. Therefore haemorrhages often begin to occur from them many days before death. Much more commonly, however, they commence only a few days or a few hours before the fatal termination. They occur in largest numbers near the inner surface, partly because the

original proliferative and degenerative changes are there most marked, and partly merely because in that situation there are more vessels from which bleeding may occur than in other regions of the dura.
In almost every case in which haemorrhage has occurred in this way from vessels near the surface similar haemorrhages may also be found in the deeper tissues of the dura. (Specimens Nos. 54 and 85)
The intense patchy congestion of the dural vessels which is so frequently seen in association with recent haemorrhages from them is to be attributed I think solely to the weakness of the capillary walls, which become dilated by the normal blood-pressure until they fill the perivascular canals. (Fig. 13)
The blood which escapes from the vessels near the inner surface finds its way into the subdural space either by simply bursting through the wall of the perivascular canals or by passing through the normal openings which possibly exist in them for the passage of the lymph. The phenomena which follow this effusion of blood into the subdural space I have already fully gone into in the preceding subsection and need not describe again.

There are still a number of points especially relating to the microscopic appearances presented by partially or fully organised false membranes which require some further words of explanation.

A large number of the cellular elements which subdural membranes contain are endothelial in character. This fact, as already stated, was pointed out by König (113). They are proliferated cells of the endothelium of the dural and arachnoid surfaces, the most of which probably became mixed with the effused blood before it coagulated. They show more or less advanced degenerative changes. (Figs. 28, 29, and 30). A distinct layer of endothelial cells spreads over the arachnoid aspect of the false membrane,- a fact to which I have already referred.

Haemorrhages occur with great frequency from the new vessels. They are I think sufficiently accounted for by the cow organisation of the false membrane and the enfeebled state of the whole vital processes of the patient. Fatty changes undoubtedly occur in the vessels (specimen 23), and many of the haemorrhages may be due to them, but they do not as a rule occur to a very marked degree, and many of the vessels may be seen to have ruptured which do not give any fatty reaction. In addition to these distinct haemorrhages in the intervascular areas, in almost every organised false membrane, isolated red

blood corpuscles are to be seen scattered about, without any apparent rupture of the neighbouring vessel wall. (Fig. 8)

Most organised false membranes contain copious haematoidin granules of an orange or brownish yellow colour. (Fig. 8) They give rise to the rusty appearance which is so prominent a feature in many cases. I have only seen this substance in these membranes in the amorphous form, and never as rhombic plates. Its development from red blood corpuscles can be studied in many preparations, and the process is of considerable interest. My observations on the point have inclined me to the opinion that its formation is always the result of leucocyte action. The mere fact that leucocytes may be seen in abundance with entire red corpuscles, disintegrating red corpuscles or these granules in their protoplasm does not, of course, prove this contention (Figs. 34 and 35.) But the following considerations must, I think, carry with them much weight. I have frequently observed in recent blood effusions into the subdural space many leucocytes containing haematoidin granules, together with others filled with entire or disintegrating red corpuscles, while not a single haematoidin granule could be seen outside of leucocytes. In the same clot granular débris produced from disintegrating red corpuscles

was in great abundance, and since it is impossible to believe that the leucocytes could show a selective action and pick out all the haematoidin granules from among this débris I am forced to the conclusion that the haematoidin must have developed within the leucocytes from some of the granular débris or entire red corpuscles which they could be seen to be taking up. I think that this may explain the curious fact that in organised subdural membranes, while the extravasated red blood corpuscles are scattered throughout the intervascular areas, the haematoidin granules become aggregated just outside of the vessels. (Fig. 8.) I suggest that what has occurred is that leucocytes have taken up red corpuscles, or the débris that results from their disintegration, and changed them into haematoidin. They have then, as it were, attempted to return to the circulation with their load, but finding it impossible to carry it through the capillary wall they have left it behind them at the side of the vessel. Interesting examples of leucocytes containing normal or disintegrating red corpuscles and haematoidin granules may be seen in specimens No 116 and 138. These haematoidin granules are often spoken of as if they were evidence only of old blood effusions. This is certainly quite a mistake. They may be found in quite recent

effusions. (See specimen No. 170)

In conclusion I wish to say a mere word about each of the three theories which have been advanced by previous writers to account for the formation of subdural membranes.

How far the haemorrhagic theory is true, and to what extent its advocates have been unconscious of the profound morbid changes which occur in the dura, must appear quite evident from what I have already said.

The inflammatory theory is I maintain, entirely disproved by the phenomena which are revealed by superficial horizontal sections of the dura mater. These show that the true subdural membrane formation of the insane is attended by degenerative changes in the tissues of the dura and not by an inflammatory process. The cellular infiltration which the advocates of this theory have pointed to in support of their views is clearly due to proliferation of endothelial cells and not to exudation of leucocytes, which are rarely to be observed in large numbers; and I have, I think, given sufficient proof that all the appearances presented by fibrinous membranes are merely those of blood clots, frequently with numerous endothelial cells added.

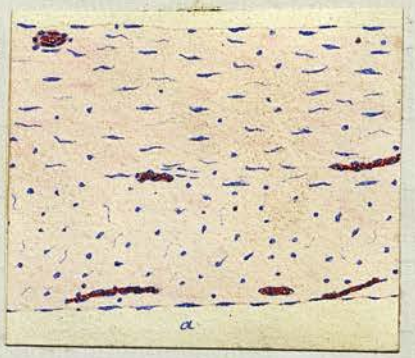
Regarding G. M. Robertson's dry-cupping theory, I doubt very much if he himself would uphold it in

the face of the microscopic evidence of dural degeneration that can be produced, and of which he was unaware. For my own part I feel satisfied that no such factor is needed to account for the morbid phenomena attending the development of subdural membranes. The whole question of pressure changes in the cranial cavity consequent upon movements of the brain and the normal mechanism by which an equilibrium is maintained is one which at present we can only theorise about. Further accurate experiments are needed upon the subject before we can have a basis of solid facts regarding it from which we can argue about the bearing of this tendency to pressure changes upon questions of intracranial pathology.

It may still be asked, What is the cause of the degenerative changes in the dural tissues which lead to the formation of subdural membranes in the insane? This is a question which at present it is impossible to answer definitely. But this much is clear, that this dural degeneration constitutes only one of a whole series of morbid changes which are specially apt to affect the coverings of the brain in the insane. Among these are to be included from within outwards, milkiness and thickening of the pia-arachnoid, this morbid change in the dura which is primarily of the same nature, thickening and

other changes in the cranial bones, addhesion of the scalp to the pericranium, degeneration of the ear cartilages followed by development in them of new vessels the rupture of which produces haematoma auris, and lastly, coarseness and thickness of the hair. Dr Middlemass and I have suggested, in the papers in the Edinburgh Medical Journal already referred to, that they may all be regarded as in part at least due to an abnormal trophic condition which is in some way reflected upon the surrounding tissues by the morbid energising of the subjacent brain which constitutes insanity.

1.



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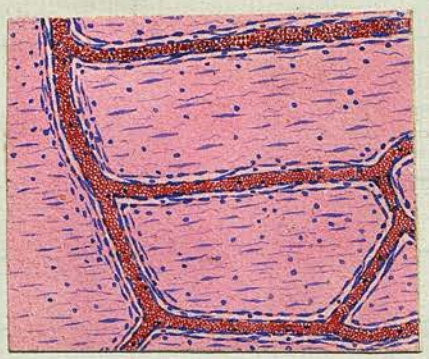

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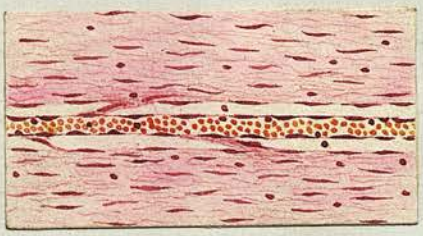

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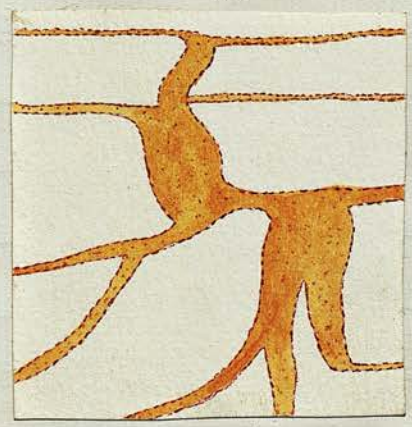

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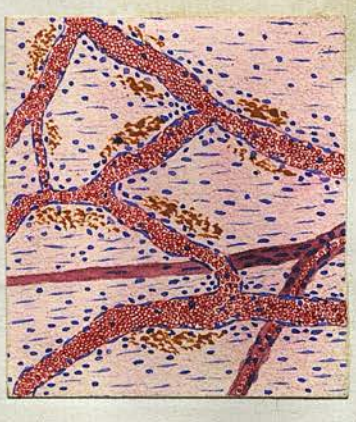

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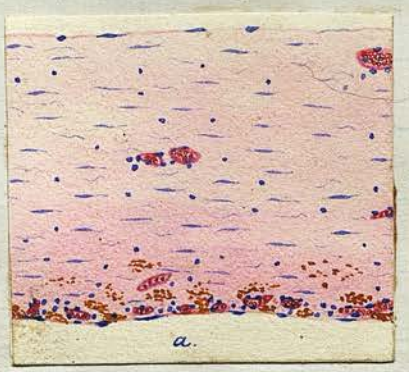

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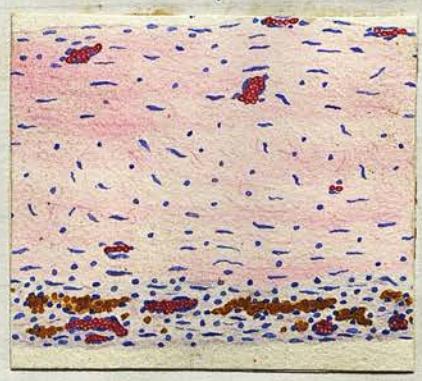

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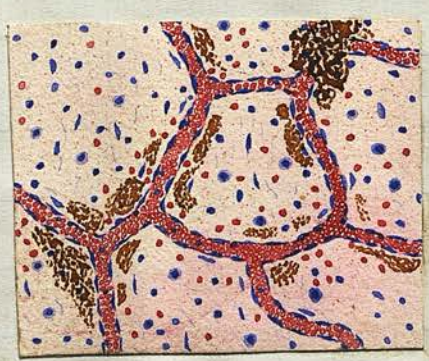

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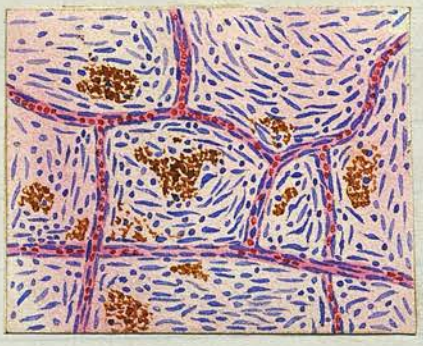

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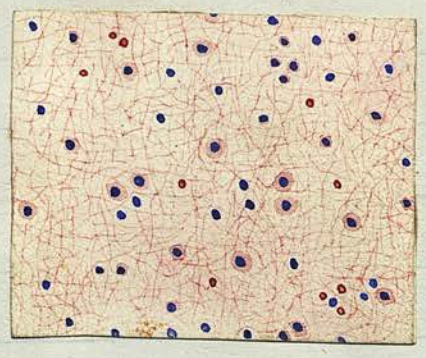

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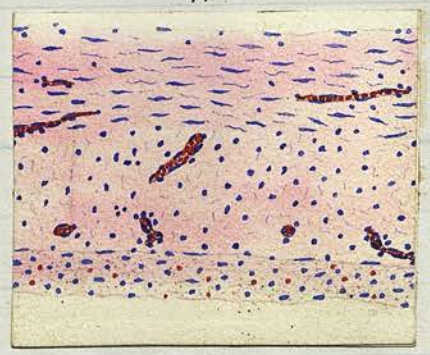

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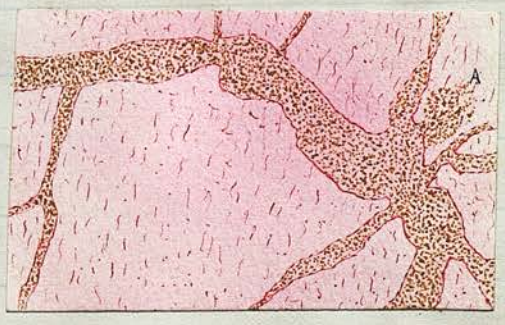

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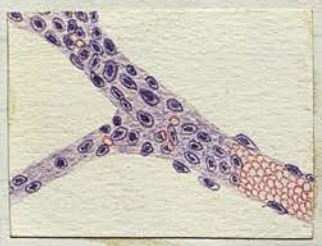

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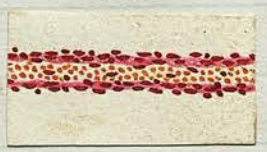

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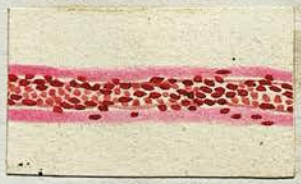

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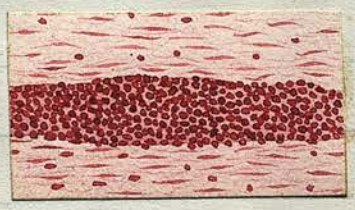

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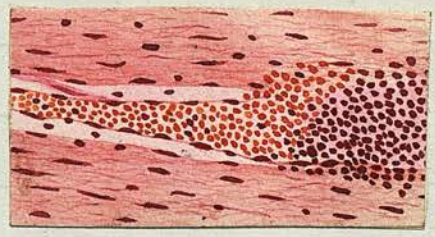

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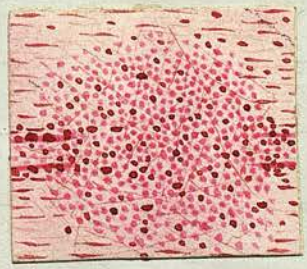

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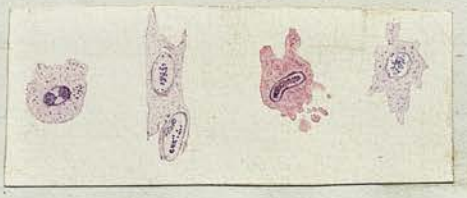

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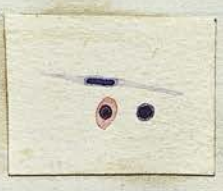

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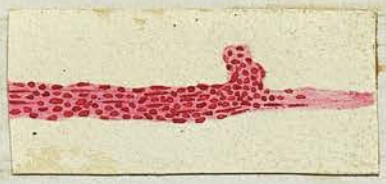

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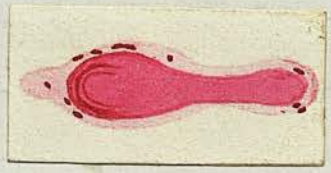

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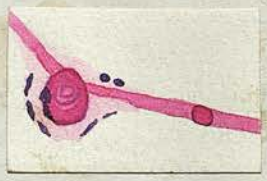

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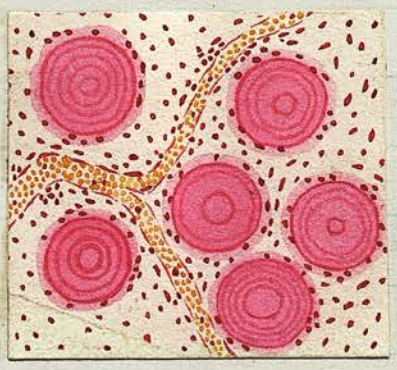

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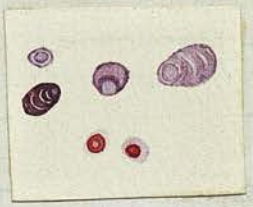

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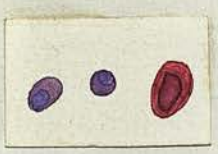

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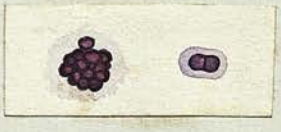

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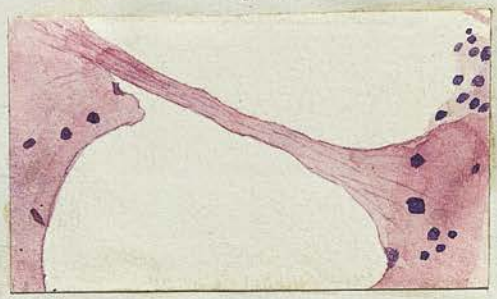

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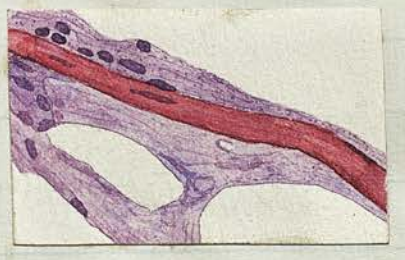

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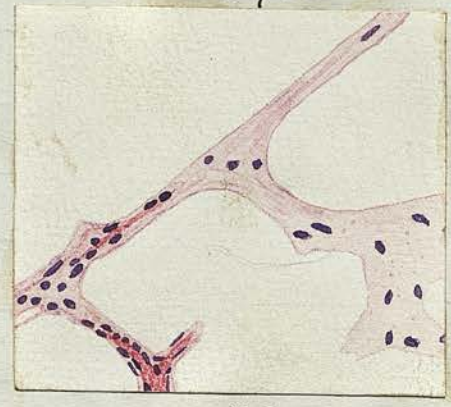

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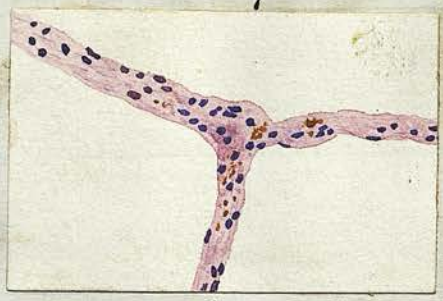

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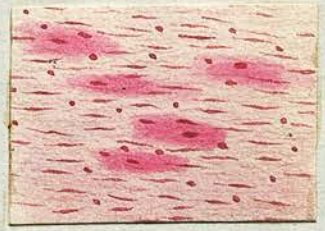

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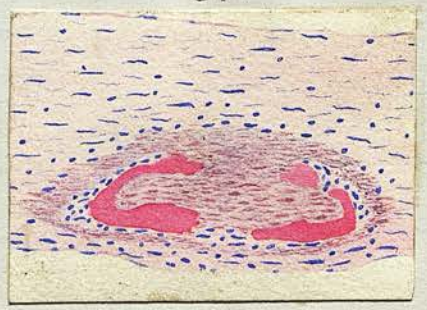

DESCRIPTION OF THE ILLUSTRATIONS.

Fig. 1. - Transverse section of normal dura mater The letter a indicates the inner surface. Haem. and Eos. (x 300).

Fig. 2. - Superficial horizontal section of inner surface of normal dura mater, showing small arteriole and capillaries with perivascular canals. H. & E. (x 450).

Fig. 3. - Superficial horizontal section of inner surface of normal dura mater, showing capillary and perivascular canal. Alum carmine and eosine. (x 450).

Fig. 4. - Ampullary dilatation of superficial capillaries of dura mater, as seen in a superficial horizontal section. Micro. Spec. No.4. Alum carmine and eosine. (x100).

Fig. 5. - Sup. hor. sect. of inner surface of dura, from a case of general paralysis (No.1), showing obliterated original capillaries and new vessels on the surface. H. & E. (x 450).

Fig. 6. - Trans. sect. of dura mater from a case of dementia, showing increase in number of capillaries near surface and deposit of haematoidin granules. H. & E. (x 300).

Fig. 7. - Trans. sect. of dura mater from a case of general paralysis (No.2), showing delicate organised subdural membrane. H. & E. (x 300).

Fig. 8. - Horizontal view of false membrane shown in previous figure. H. & E. (x 450).

Fig. 9. - Horizontal view of portion of false membrane from same case, showing fibrous development. H. & E. (x 450).

Fig.10. - Horizontal view of fibrinous subdural membrane. Case No.15. H. & E. (x 450).

Fig.11. - Trans. sect. of dura mater and false membrane from same case. H. & E. (x 300).

Fig.12. - Sup. hor. sect. of inner surface of dura mater, showing haemorrhage from new capillaries. Case No.5. H. & E. (x 450).

Fig.13. - Sup. hor. sect. of sphenoidal dura from case No.5, showing varicose dilatation of capillaries, with rupture at A. Alum Carmine and Eos. (x 60).

Fig.14. - Aggregation of endothelial cells upon superficial capillary. Micro. spec. No.65. H. & E. (x 600).

Fig.15. - Capillary of inner surface of dura, showing thickening of its wall, and proliferation of the endothelial cells of perivascular canal. Case No.1. Alum Carmine and Eosine. (x 450).

Fig.16. - Do. showing thickening of wall only.

Fig.17. - Horizontal view of superficial capillary of dura, showing hyaline sheath, Case No.17. H. & E. (x 450).

Fig.18. - Do. Do. Case No.1. Alum carmine and eosine. (x 450).

Fig.19. - Horizontal view of superficial capillary of dura, showing thickening of its wall and deposit of haematoidin granules in perivascular canal. Case No.1. Alum carmine and eosine. (x 450).

Fig.20. - Do. showing dense aggregation of cells in perivascular canal. Case No.3. Al. car. and Eosine. (x 450).

Fig.21. - Do. Do. with rupture of vessel behind obstruction. Case No.4.

Fig.22. - Horizontal view of superficial capillary of dura, showing thickening of its wall and recent haemorrhage. Case No.4. Al. car. and eos. (x 450).

Fig.23. - Horizontal view of superficial capillary of dura, showing mass within it blackened by osmic acid. Case No.4. Osmic acid and picrocarmine. (x 450).

Fig.24. - Horizontal view of endothelial cells of surface of dura, showing granularity and commencing disintegration of cell plate, and early nuclear changes. Case No.9. H. & E. (x 600).

Fig.25. - View of surface of dura mater from same case, showing proliferated endothelial cells with similar degenerative changes. H. & E. (x 600).

Fig.26. - Endothelial cells in fibrinous membrane. Case No.2. Micro. spec. No.24. H. & E. (x 600).

Fig.27. - Endothelial cells of inner surface of dura, showing vacuolation. Case No.2. H. & E. (x 600).

Fig.28. - Endothelial cells in false membrane showing advanced degenerative changes,-vacuolation and vitreous degeneration. Case No.2. H. & E. (x 600).

Fig.29. - Degenerated endothelial cells in false membrane. Case No.2. H. & E. (x 600).

Fig.30. - Do. Do.

Fig.31. - Horizontal view of two endothelial cells of inner surface of dura, showing slight granular change in cell plate. Case No. 11. H. & E. (x 600).

Fig.32. - Leucocytes and spindle cell in subdural membrane. Case No.11. H. & E. (x 600).

Fig.33. - Leucocytes in subdural membrane. Case No.2 H. & E. (x 600).

Fig.34. - Do. containing haematoidin. From same case.

Fig.35. - Do. containing red blood corpuscles. From subdural membrane. Case No.12. H. & E. (x 600).

Fig.36. - Hyaline rod seen in superficial horizontal section of dura. Case No.1. H. & E.(x500)

Fig.37. - Do. in false membrane. Case No.18. H. & E. (x 500).

Fig.38. - Degenerated new capillary forming hyaline rod studded with cells. Sup. hor. sect. of dura. Case No.1. Al. car. & Eos. (x 450).

Fig.39. - Hyaline rod probably developed from degenerated new capillary. From same case as preceding.

Fig.40. - Hyaline rod apparently contracting into a concentric body. Same case as preceding. Micro. spec. No.9.

Fig.41. - Do. Do. Micro.spec. No.14.

Fig.42. - Do. Do. Micro. spec. No.11.

Fig.43. - Hyaline rod in subdural membrane apparently developing into concentric bodies. Case No.18. Micro. spec. No.110. H. & E. (x 500).

Fig.44. - Developmental form of concentric body. Case No.18. Micro. spec. 111. H. & E. (x 500).

Fig.45. - Do. Do. al. car. & Eos. (x 500).

Fig.46. - Do. Do. Micro. spec. 111. H. & E. (x500)

Fig.47. - Concentric body in a fibrinous false membrane. Case No.15. Haem. (x 450).

Fig.48. - Concentric bodies on surface of dura. Silver preparation. Case No.16. Micro. spec. No.97. (x 500).

Fig.49. - Group of concentric bodies in superficial horizontal section of dura. Case No.1. Micro. spec. No.14. Al. car. & eos. (x 450).

Fig.50. - Concentric bodies of the starch grain form. Case No.12. H. & E. (x 500).

Fig.51. - Do. in deep horizontal section of dura near middle meningeal artery. Case No.9. Micro. spec. No.68. H. & E. (x 500).

Fig.52. - Mulberry bodies. Case No.10. Micro. spec. No.71. H. & E. (x 500).

Fig.53. - Mulberry body. Case No.11. H. & E. (x 500).

Fig.54. - Delicate film on surface of dura (seen in superficial horizontal section) undergoing contraction. Case No.1. Micro. spec. No.7. H. & E. (x 500).

Fig.55. - Do. Do. forming fenestrated membrane. Case No.3. Micro. spec. No.40. H. & E. (x 500).

Fig.56. - Degenerated new capillary in contracting fibrinous film. Case No.6. Micro. spec. No.58. H. & E. (x 500).

Fig.57. - Contracting fibrinous film on surface of dura furnishing sheath to degenerated new capillary which is seen to be continuous with superficial vessel of dura. Case No.1. Micro, spec. No.10. H. & E. (x 500).

Fig.58. - Contracting fibrinous film on surface of dura and degenerating new capillary, to one side of which there is attached a hyaline cord developed from the film. Case No.1. Micro. spec. No.7. H. & E. (x 500).

Fig.59. - Degenerated new capillary on the surface of the dura, showing haematoidin granules within it. It is probably covered by a sheath formed by the fibrinous film into which it grew contracting round it. Case No.1. Micro. spec. No.7. H. & E. (x 500).

Fig.60. - Sup. hor. sect. of dura, showing deeply stained and slightly granular patches in the fibrous tissue. Case No.20. Al.car. and eos. (x 450).

Fig.61. - Trans. sect. of dura, showing area of degenerative change. Micro. spec. No.15. H. & E. (x 450). For full description see under case No.1.

MICROSCOPIC SPECIMENS.

A box containing 205 microscopic specimens is produced along with this thesis. As each specimen is referred to in consecutive order in the note on the normal structure and in the descriptions of the cases, it is not thought necessary to give an inventory of them.

On the labels the following contractions are employed:-

T. S. = Transverse section.

S. H. S. = Superficial horizontal section.

H. & E. = Haematoxylin and eosine.

A. C. & E. = Alum carmine and eosine.

Particular spots to which attention is directed are marked with an ink line on the under surface of the slide leading up to them. When more than one spot is marked the indicators are distinguished by the number of cross lines. Whenever a special spot in a specimen is referred to in the course of the descriptions of microscopic appearances, the mark which points to it is affixed to the number.